The Journal of
Bone and Joint Surgery

EDITORIALS AND ANNOTATIONS

CRUSH INJURIES OF THE CHEST

It is not easy to define the pathological sequelae of crush injury of the chest. The terms " stove-in chest " and " flail chest " are used when a patient has sustained fractures of many ribs and there is paradoxical movement of the chest wall. But sudden and severe compression may produce dyspnoea without fracture of the ribs and without paradox. Moreover, even when there are rib fractures with paradoxical movement of the chest, the paradox may be evidence of increased respiratory effort from other causes rather than the source of the respiratory embarrassment. These causes of respiratory insufficiency include obstruction of the tracheo-bronchial tree, collapse, contusion, oedema or infection of the lung, reduced diaphragmatic movement, reflex inhibition of respiratory movement from pain, and functional disorganisation of the neurones controlling breathing.

The mechanisms of paradox are well understood. If a segment of the chest wall is flail the degree of paradoxical movement varies with respiratory effort. In eupnoea it is slight or absent; in dyspnoea it is usually marked and limits the normal range of ventilation; but always the mobility of the injured part depends on the underlying cause of the dyspnoea. Thus adequate clearance of the trachea and bronchi, pleural drainage and relief of pain may in the first instance abolish dyspnoea and so control the paradox. Nevertheless it will reappear if other complications such as lung oedema, infection, bronchospasm, abdominal distension, fever or uraemia develop in the hours or days after injury. In patients with chronic lung disease, or in those who suffer severe damage to the lung as well as the chest wall at the time of injury, dyspnoea and paradox persist. In such circumstances paradox becomes important in the genesis and maintenance of respiratory insufficiency and requires specific treatment.

In emergency management of patients with crush injury of the chest, non-respiratory factors may be important. Multiple injuries with complex physiological derangement of the central nervous and circulatory systems make it difficult to determine the source of the dysfunction or indeed to separate cause from effect. For example, when a patient admitted to the casualty department is unconscious it may be the result of head injury, respiratory insufficiency, or both. Failure to distinguish the cause may be disastrous. If the patient has severe circulatory failure from respiratory or cardiac causes, treatment with massive blood transfusion has its hazards. Some of these difficulties can be overcome by treating respiratory troubles first (Barrett 1960, Griffiths 1960). In patients with head injury, respiratory insufficiency prejudices the outlook (MacIver, Frew and Matheson 1958); urgent, competent and sometimes prolonged management of respiration is demanded, and this is not made easier by the restlessness that is so usual when consciousness returns.

There may also be acute renal failure with three further hazards to the patient whose chest is injured. The onset of serious hyperkalaemia may be rapid, particularly if there is lung infection with respiratory acidosis. Metabolic acidosis from renal failure demands respiratory compensation which may be beyond the patient's power. The neurological complications of

uraemia are aggravated by hypoxia and respiratory acidosis. This raises the question whether prophylactic dialysis should be used for seriously injured patients as advocated by Teschau and Baxter (1960).

In promoting ventilation some forms of treatment are now generally accepted: clearance of secretions from the trachea and bronchi, re-expansion of the lungs by pleural drainage, relief of pain, and institution of the tracheostomy regime (Carter and Giuseffi 1951, Hulman 1957). In most patients these measures are enough, though carefully recorded observations of changes in pulse, blood pressure, the pattern of breathing and levels of consciousness should be maintained for several days because a relapse into respiratory inadequacy is not uncommon and may be insidious. If at any time there is doubt as to whether or not the ventilation is adequate, a short trial period of hand inflation using a Waters' carbon dioxide absorption unit will resolve the problem. Improvement in the patient's condition will be manifest and often he will indicate his wish to continue with it.

In a few patients dyspnoea and paradox persist or return because of complications, and steps must then be taken to improve respiratory function. Two methods are now available: stabilisation of the chest wall by surgery, or control of breathing by intermittent positive pressure ventilation—but opinion is sharply divided on their respective merits.

The advantage of surgical stabilisation of the flail segment has often been stressed (Jones and Richardson 1926, Butler 1938, Williams 1948, Jaslow 1946, Proctor and London 1955, Heroy and Eggleston 1951, Ritter and Kaye 1944, McKim 1943, Coleman and Coleman 1950, Bickford and Grant 1956, Robin 1960, Virshup 1960 and Sillar 1961). It is to be remembered that many of these reports were written before 1956 when there was no alternative treatment. Moreover some are individual case records and, perhaps significantly, only of patients that survived. Three papers record small series of cases. Coleman and Coleman (1950), Bickford and Grant (1956) and Sillar (1961) together report twenty-six cases with five deaths. Criteria of what constituted a severe crush injury of the chest varied greatly, and today many such patients would have been treated successfully by tracheostomy alone. Nevertheless a few are examples of remarkable achievement. The essence of the matter seems to be whether surgical methods can ensure sufficient stabilisation of the chest wall to restore normal ventilation, can this be done in all types of injury to the chest wall and is it wise to undertake what may be a prolonged and difficult operation in seriously injured people, the outcome of which in terms of respiratory function may be doubtful? Sellors (1961) and Windsor and Dwyer (1961) expressed serious doubts and we agree.

The alternative method is to take over control of the ventilation and be sure that the requirements of stability and good ventilation are met. This is achieved first by inducing apnoea, and then by maintaining gaseous exchange by a mechanical ventilator. It must be emphasised that because spontaneous breathing is abolished, triggering mechanism in the ventilators are unnecessary. Furthermore, a negative phase is undesirable because it will produce paradox. We have found the Newcastle and Barnett machines adequate in such management provided that treatment is started early, before congestion, oedema and infection are well established in the lungs.

Avery, Mörch and Benson (1956) discussed these principles and reported a case treated successfully. Boyle, Gallie and Murray (1957) reported two patients with crush injury of the chest, one treated by traction on the chest wall, and the other by positive pressure ventilation: both patients died. Recently this method has been advocated in three papers (Griffiths 1960, Fairley and Chambers 1960, Windsor and Dwyer 1961). Griffiths reported six cases with three deaths, but since publication ten other patients have been treated without death. Fairley and Windsor recorded fifteen cases with two deaths, making a total in the three series of thirty-one patients so treated, with five deaths. All had sustained severe crush injury of the chest; some were very aged and with chronic lung disease or multiple injuries. Of the five deaths reported, three were caused by such delay in starting treatment that advanced pulmonary

changes made ventilation of the lungs impossible. The other two deaths were from acute asphyxia which could have been avoided (Griffiths 1960).

Treatment of severe crush injuries of the chest by this method has many advantages quite apart from avoiding the risks of big operations in very ill patients. It does more than patch up the chest wall; it corrects the respiratory insufficiency, and in so doing it abolishes paradox. It maintains inflation of the lung and thus provides a form of internal splinting of the chest wall. The minute volume can be adjusted to meet increasing ventilatory requirements such as may arise with abdominal distension, infection, fever, restlessness and acute renal failure. Of course the technique is not without its hazards. The creation and maintenance of such an unphysiological mechanism as controlled respiration for several weeks is a critical undertaking, requiring special knowledge and facilities. It must be supervised constantly by an experienced nursing and medical staff, and it is best carried out in a special unit with a single person in charge who undertakes full responsibility for the respiratory problem. Nowhere in medicine is it truer to say that " too many cooks spoil the broth." Such a unit should be attached to a wider organisation dealing with the whole problem of trauma.

Barber, Chambers, Fairley and Woolf (1959) have discussed the establishment of a unit designed to treat all forms of respiratory failure. This, we believe, is the only way by which to reduce the high mortality associated with injuries to the chest. Until such units are set up surgical fixation of the chest wall may remain the only treatment possible or practicable; but its justification will be based on expediency rather than merit. H. W. C. GRIFFITHS.

REFERENCES

AVERY, E. E., MÖRCH, E. T., and BENSON, D. W. (1956): Critically Crushed Chests. *Journal of Thoracic Surgery*, **32,** 291.

BARBER, H. O., CHAMBERS, R. A., FAIRLEY, H. B., and WOOLF, C. R. (1959): A Respiratory Unit. The Toronto General Hospital Unit for the Treatment of Severe Respiratory Insufficiency. *Canadian Medical Association Journal*, **81,** 97.

BARRETT, N. P. (1960): Early Treatment of Stove-in Chest. *Lancet*, **i,** 293.

BICKFORD, B. J., and GRANT, A. F. (1956): Surgical Management of the " Stove-in " Chest Injury. *Annals of the Royal College of Surgeons of England*, **19,** 371.

BOYLE, A. K., GALLIE, J. R., and MURRAY, D. B. (1957): Crush Injury of the Chest. A Report on Two Cases. *Anaesthesia*, **12,** 453.

BUTLER, E. (1938): Injuries of the Chest and Abdomen. *Surgery, Gynecology and Obstetrics*, **66,** 448.

CARTER, B. N., and GIUSEFFI, J. (1951): Tracheotomy, a Useful Procedure in Thoracic Surgery, with Particular Reference to its Employment in Crushing Injuries of the Thorax. *Journal of Thoracic Surgery*, **21,** 495.

COLEMAN, F. P., and COLEMAN, C. L. (1950): Fracture of Ribs—A Logical Treatment. *Surgery, Gynecology and Obstetrics*, **90,** 129.

FAIRLEY, H. B., and CHAMBERS, R. A. (1960): The Management of the Patient with Respiratory Insufficiency. *Canadian Anaesthetists' Society Journal*, **7,** 447.

GRIFFITHS, H. W. C. (1960): Crush Injuries of the Chest. *Journal of the Royal College of Surgeons of Edinburgh*, **6,** 13.

HEROY, W. W., and EGGLESTON, F. C. (1951): A Method of Skeletal Traction Applied through the Sternum in " Steering Wheel " Injury of the Chest. *Annals of Surgery*, **133,** 135.

HULMAN, S. (1957): Tracheotomy for Flail Chest. *Lancet*, **i,** 454.

JASLOW, I. A. (1946): Skeletal Traction in the Treatment of Multiple Fractures of the Thoracic Cage. *American Journal of Surgery*, **N.S. 72,** 753.

JONES, T. B., and RICHARDSON, E. P. (1926): Traction on the Sternum in the Treatment of Multiple Fractured Ribs. *Surgery, Gynecology and Obstetrics*, **42,** 283.

MCKIM, L. H. (1943): A Method of Fixation for Fractures of the Sternum. *Annals of Surgery*, **118,** 158.

MACIVER, I. N., FREW, I. J. C., and MATHESON, J. G. (1958): The Role of Respiratory Insufficiency in the Mortality of Severe Head Injuries. *Lancet*, **i,** 390.

PROCTOR, H., and LONDON, P. S. (1955): The Stove-in Chest with Paradoxical Respiration. *British Journal of Surgery*, **42,** 622.

RITTER, H. H., and KAYE, B. B. (1944): Trauma of the Thorax. *American Journal of Surgery*, **N.S. 64,** 191.

ROBIN, G. C. (1960): Sternal Traction for the Flail Chest. *Lancet*, **i,** 26.

SELLORS, T. Holmes (1961): The Management of Chest Injuries. *Thorax*, **16,** 1.

SILLAR, W. (1961): The Crushed Chest. Management of the Flail Anterior Segment. *Journal of Bone and Joint Surgery*, **43–B**, 738.

TESCHAU, P. E., and BAXTER, C. B. (1960): Comparative evaluation of Macneil-Collins twin coil and rotating drum dialysers, ultrafiltrates, chronic vessel cannulation and regional heparinization, with special reference to prophylactic hemodialysis. *American Association of Artificial Organs*, **6**, 76.

VIRSHUP, M. (1960): The Anteriorly Crushed Chest or "Steering-wheel Injury." *Journal of Thoracic and Cardiovascular Surgery*, **39**, 579.

WILLIAMS, M. H. (1948): Severe Crushing Injury to the Chest. *Annals of Surgery*, **128**, 1,006.

WINDSOR, H. M., and DWYER, B. (1961): The Crushed Chest. *Thorax*, **16**, 3.

THE PROBLEM OF THE RELAPSED CLUB FOOT

Club foot in infants is one of the conditions described by Hippocrates, and he was clearly familiar with the basic principles revived by Kite thirty years ago; but the problem of the relapsed club foot has been tackled only comparatively recently. W. J. Little in 1839 described his success with tenotomies and careful splinting, and it was only in the second half of the nineteenth century that a "complete and permanent cure" was found by William Adams with a mutilating operation involving arthrodesis of the major joints in the foot. It soon became apparent that the results of such drastic surgery were disappointing and that further relapse could occur. Since then a solution has been sought by many different surgeons but the multiplicity of operations is an indication that the problem has not been solved. Each operation has had success—partial or complete—claimed for it, but the visitor to orthopaedic centres still finds many differing views, and their out-patient departments provide ample evidence that we are a long way from finding a satisfactory cure for all club feet.

The difficulties that face any worker in this field are great, among them the uncertain etiology and the doubt as to the site of the primary deformity, a difficulty which is increased by the fact that secondary changes are well established by the time the child is born. If it is assumed that the primary deformity is in the midtarsal joint the complexity of the secondary deformities raises further problems, and these may involve most of the structures below the knee; so although the name talipes concentrates attention upon the foot, account has to be taken of abnormalities well above that level. Add to this the variations in the severity of the deformity and in the response of the individual foot to treatment, and the surgeon may be faced with a problem that defeats his skill, persistence and ingenuity.

In the relapsed club foot the two stages of securing correction first and then maintaining it are sometimes confused, but they should be clearly distinguished, at least in the surgeon's mind. No correction can be considered complete until there is full passive correction, and no foot can be considered secure from relapse until the child has a plantigrade foot and until muscle balance has been restored.

The two papers in this number of the Journal show a different approach to the problem. Singer postulates full passive correction before any tendon transplant is undertaken, and there is no doubt that he is right on this point; but the difficulty arises when, as is sometimes the case, this is found to be impossible of achievement. Tendon transfer as an isolated operation has only a limited application. By experience Singer has found the disadvantages of using tibialis anterior to restore muscle balance, and his results with tibialis posterior are encouraging. If the tendon transfer were performed at the same time as a soft-tissue correction it might well increase the scope of the operation.

Dillwyn Evans assumes that the primary displacement occurs in the midtarsal joint, a complex series of secondary and adaptive changes resulting in the familiar club foot deformity, and he bases his treatment upon that hypothesis. He stresses the importance of dividing the tight structures at the inner side of the foot and of reducing the subluxated head of the talus into its socket, but he has found that this is inadequate until the relative over-lengthening of the outer column of the foot has been corrected and stability restored by arthrodesis of the

calcaneo-cuboid joint. Sufficient bone must be resected to permit lateral rotation of the navicular bone, so bringing the first metatarsal bone into line with the talus. He claims that by this means the other deformities can be controlled, and that the relative lengths of the medial and lateral columns of the foot are partly responsible for the valgus and varus inclinations of the heel. His statement that an equino-varus deformity can be produced by lengthening of the calcaneum is also of interest.

This operation deserves careful consideration. It is not just one more attempt to deal with the relapsed club foot. It is based upon a hypothesis that is widely, if not universally, held; it has the merit of correcting the deformity without doing extensive damage and of leaving the child with a plantigrade, well balanced foot. If it can be shown that the operation is effective without a tendon transfer, it will represent a great advance in the surgery of this difficult field.

A. T. FRIPP.

REFERENCES

ADAMS, W. (1866): *Club-Foot: its Causes, Pathology, and Treatment.* London: John Churchill and Sons.

HIPPOCRATES: *Genuine Works of Hippocrates Translated by Francis Adams*, (1849), p. 632. London: Sydenham Society.

KITE, J. H. (1935): The Treatment of Congenital Club-Feet. *Surgery, Gynecology and Obstetrics*, **61**, 190.

LITTLE, W. J. (1839): *A Treatise on the Nature of Club-Foot and Analogous Distortions.* London: W. Jeffs, S. Highley.

THE SCIENTIFIC SURGEON

Presidential Address: British Orthopaedic Association, Manchester, April 14, 1961

H. J. SEDDON, LONDON, ENGLAND

In the development of orthopaedics in this country there is much of which we may be proud. We have made many contributions to our craft, and have pioneered the development of regional hospitals and their peripheral clinics. There is still more to be done—ubiquitous accidents pose new problems of organisation and treatment—indeed, more than enough to occupy all our efforts and skill. The result is that there is all too little time to spare for studying the phenomena of injury and disease, or even to examine current methods of treatment with calm detachment. Good practice is to some extent the enemy of science. This is not peculiar to orthopaedics; it obtains in every branch of medicine and in many other kinds of human endeavour. So I am going to say something about research in relation to orthopaedics.

Discourses on research or on the art and science of medicine have been given with dismal frequency. Two features appear fairly regularly. The orator often betrays an uneasy consciousness of the apparent conflict between the scientific advancement of medicine and its practice. In a series of addresses, remarkable alike for their erudition and for the beauty of their language, Walshe (1948, 1950, 1956) set out to prove that there is, after all, no conflict. I wish I could be convinced. Another speaker, Arnott (1955), denounced the empiricism of medicine root and branch and thought the whole business should be cleaned up. The other and more interesting feature is the light these addresses often throw on the mind of the orator. They tend to be an *apologia pro vita sua*. I am aware of this risk in speaking to you today.

Let me say straight away that the conflict between science and practice is there; we must face it. We are now in the realm of ideas. But different ways of thinking come from different kinds of men, and there are many such in medicine. Let me describe four types to you by the cruel but evocative art of caricature, a sort of Hogarth portrait gallery.

1. *The Scientist*. He is often pictured as bearded, wearing a leather-bound tweed jacket, a regular reader of the *New Statesman*, a violinist, a connoisseur of wines. He may seem to lack humanity; one man with whom I once worked habitually referred to the patient as " the preparation." His Olympus is the Royal Society; his apotheosis the Nobel Prize.

2. *The Consultant*. Bernard Shaw's Cutler Walpole. He exudes charm, is impeccably dressed, and his waistcoat is double-breasted. The absence of any original idea beneath his brow has kept it unfurrowed. An elegant way of life and a certain place in society are regarded as indispensable. His Valhalla is the Council of the College of Surgeons; he even dreams of becoming President, ruler of that great empire in which the concrete never sets. He does not understand that without certain higher qualities he can never get there.

3. *The Missionary*. A very different type. He practises medicine because somewhere deep inside him is a real devotion to his fellow men. He may come from a great family of doctors. He is most conscientious though he may do the wrong thing, for the reason that he gives so generously of his time to good causes that he can never quite get round to becoming competent.

4. *The Administrator*. The organiser, the committee man, the negotiator. A splendid speaker, most useful in his way, indeed indispensable. What he really loves is power, what he covets is public recognition.

These are crude, harsh sketches, though occasionally one finds these people in real life. Most of us are a mixture and this is what makes us such fascinating creatures; but one

characteristic often predominates. Today I am concerned only with the scientist and the capable practitioner because they represent the Science and the Art of Medicine. We must not fight shy of the conflict between them; it is inevitable and it can be healthy. Freedom to differ is essential for the development of the mind. But with it—and this is true of nations, social and professional groups, and of individuals—there must be tolerance. What counts is the breadth of a man's mind and sympathies.

The full-time scientist leads an exacting life, but, if he is a wise man, it is designedly simple. The pursuit of learning has long been associated with the cloister. This is illustrated most graphically by the colleges of our ancient universities where, even today, a man may retire into his cell and devote himself to original work. The clinician, by contrast, lives in the bustle of the market place. The world presses round him; his patients see to that. Yet there are temptations common to the don and to the hospital consultant. The allurements of administration can break through the stoutest college gates and many a promising young scholar has become a university busybody. If the clinician is to make use of the unique opportunities for learning which the practice of medicine affords, he must create his own retreat, and limit his outside commitments and distractions. His way of life must be simple, otherwise he will have no time to think.

In medical practice some environments are more conducive than others to research; some carry special obligations.

1. At the frontiers of medicine, as we see them in young territories overseas, the pressure of routine work is overwhelming; a man has to confine himself to what he can do most profitably with limited resources and perhaps no skilled assistance. If he finds that his natural curiosity is leading him into some sort of investigation he must seek a change of environment, and fortunately this is possible. Men with ideas are at a premium.

2. In regional hospitals the orthopaedic surgeon may have to work alone or with one other colleague. These men have to be good all-rounders and to be shrewd in the selection of the methods they employ for the conditions that demand most of their attention. If they are wise they do not dabble with rarities but decant them to the nearest big centre. If the grind of work does not exhaust them they may make excellent contributions in the clinical field. One of the most striking examples was Brittain of Norwich. These men are invaluable because they see orthopaedics as a whole.

3. The undergraduate teaching hospital. This is part of a seat of higher education and by definition research is one of its activities. A man can be a good teacher, an excellent practitioner and do no research. He will be remembered by his pupils, perhaps revered by them, but he will have failed to some extent in his destiny if he has not brought those in his charge to the frontiers of the unknown, and encouraged distrust in cant and dogma. They will see no promised land beckoning them. The surgeon on the staff of a teaching hospital must have at least a sympathy with and some understanding of research, even though his own gifts may not lead him in that direction.

4. Lastly, we come to the special hospital of which this country has a considerable number. They were founded to make provision for patients for whom the general hospitals did not adequately cater, and for the advancement of knowledge in a particular field. If the special hospital with its peculiar advantages fails to promote research, to advance knowledge, it neglects one of its purposes. Some have failed and we may find that the penalty for this is dissolution.

So much for the background. Now, may I address myself more particularly to younger members of this Association who still have their professional life before them? To you is committed a part of the future of medicine. In this audience there are a number of men who although not passionately devoted to research (if they were they would be seeking their careers elsewhere) are sufficiently endowed with curiosity and the capacity to think and work

scientifically to fit them to advance medicine. Here are a few simple observations which will repel or attract you, but to which you cannot be indifferent.

1) Endurance. Any quest for knowledge calls for hard work. Inspiration without it is like faith without works—dead, being alone.

> The heights by great men reached and kept
> Were not attained by sudden flight,
> But they, while their companions slept,
> Were toiling upwards in the night.
>
> Longfellow, *The Ladder of St Augustine*

This, you may think, savours too much of Samuel Smiles. Perhaps so, but the Victorian virtues are not to be despised. 2) A simple way of life. My favourite example in British surgery is Lord Lister. If you read Godlee's biography carefully you may come to the conclusion that has been forced on me—I put it to you very humbly—that he was not a supremely clever man. It was simplicity and childlike honesty that marked his thoughts and work. He had to earn his living in consulting practice though he was never a brilliant surgeon. Yet, from his earliest days he had an astounding grasp of what mattered; and with it went a dogged determination to pursue those experiments and practices that placed the whole world in his debt. And a glorious harmony marked his long and fruitful life. I know men today of like character though the times do not invest them with Lister's heroic lineaments. 3) No time like the present. Research is a young man's game. Granit, the great Swedish physiologist, once told me that a man's best work is done before the age of forty-five. Apart from the springs of originality running dry, the burden becomes too heavy as the years pass. For us there is a difficulty here. In clinical medicine a certain amount of experience is necessary before the investigator can get an eye for a problem. Yet the discerning young man will often spot it and we must provide means for him to work on it while the problem is still fresh in his mind. Don't wait: time is not on your side. 4) Undue modesty. Experience counts for so much in clinical medicine that the tyro may feel seriously inhibited by his lack of it. I cannot do better here than quote that great Spaniard, Ramón y Cajal (1951): " I consider among the most doleful preoccupations of intellectual youth is undue admiration for the work of great minds . . . This excessive devotion to genius has its roots in a feeling that is a compound of justice and modesty much too commendable to be censured. Nevertheless, if it takes overweening possession of the mind of the novice, it destroys all initiative and totally incapacitates him for original investigation. Defect for defect, arrogance is preferable to diffidence; boldness measures its forces and conquers or is conquered, but excessive modesty flees from the battle and is condemned to shameful inactivity." Or more tersely, more sternly, St Paul to Timothy, " Neglect not the gift that is in thee. Let no man despise thy youth." 5) Concentration. When you have got your teeth into something, keep them there. You will have noticed how great scientists—like Adrian and Dale; in surgery, Penfield—have followed much the same type of work throughout a long and fruitful lifetime. The lists of papers I see appended to applications for senior appointments are sometimes depressing, a pot-pourri of superficial contributions on far too many subjects. When you have found an interesting line of work stick to it; don't be distracted by every other seductive idea that comes along. 6) The approach to a topic. There are distinct stages in the handling of a subject. There is the first flush of enthusiasm, when interest is aroused. Then you begin to wade through the literature and disillusionment creeps in. It seems that others have done everything. Don't be discouraged. As you ponder you will become aware of the gaps in our knowledge or of ideas long accepted as gospel but palpably unsound, and that must be challenged. Write out what you plan to do, then set to work. After five years or so you may find you are stuck;

it appears there is nothing more to be done. Turn your mind to something else for a while. Later you may return to your first love, and the chances are that you will once more find something new which you take up with an interest sweetened by long association. Before you know where you are it is only ten years before you are due to retire, and the shortness of life for a man who seeks knowledge will be brought home to you with startling urgency.
7) The sacrifice of all-round competence. There are exceptional men who excel both scientifically and in comprehensive practice in their field of surgery. But most of us with an interest in the science of surgery are compelled to limit our repertoire so far as operative work is concerned, simply because technical skill is largely a matter of practice and practice takes time. This limitation applies only to the more complex forms of treatment. It must not be tolerated in diagnosis or in our grasp of medicine as a whole. It is noticeable that scientists intensely devoted to one line of investigation often display an astonishing breadth of interests. This is a mark of the cultivated mind. But in the sort of work that you and I do it means that if we are to find time for research we must be prepared to hand over patients with certain conditions to colleagues particularly skilled in the treatment of them. This is not specialisation within a speciality: it is plain commonsense. It enables a group of surgeons, perhaps covering a wide area, to help each other forward. This is one kind of team work.

As surgeons we have certain advantages over other clinicians, for we are able to look inside the human body. The operation theatre is the greatest of all laboratories in medicine and it is ours. The trouble is that we too often approach it as if it were the stage.
a) An operation may enable us to observe phenomena or collect material which have no direct connection with the operation itself. Kellgren in this city made observations on the sensibility of articular structures during the course of operations on the knee under local anaesthesia.
b) The operation may yield information about the nature of a disorder. This is how Mandl cleared up the mystery of hyperparathyroidism: this is how we found the causes of median paralysis at the level of the carpal tunnel and of paralysis of the deep branch of the ulnar nerve.
c) The operation may be part of a planned trial to determine whether one method is better than another; here there must be the most searching documentation of pre- and post-operative conditions. But as I shall explain in a moment, this sort of thing is all too rare.
d) The operation may itself be an experiment, the aim being to achieve a result previously unattainable, or to obtain a good result more surely, more safely, more simply than before. For this the most intensive preparation is necessary, perhaps involving animal experiment, as in the recent spectacular work on the heart. Inadequate preliminary work or publication of a new method before sufficient time has elapsed for the disclosure of possible shortcomings can be disastrous. Need I do more than remind you of some of the recent work on the reconstruction of the hip joint?

Now supposing you have hit on something that looks really good; it may be a new operation, or a non-operative method of treatment. You identify yourself with it, you become a partisan, an advocate. That is fair enough—but only at first. There is quite a chance that an equally competent surgeon a hundred miles away will claim that he can get as good or even better results by a quite different method. What happens? The secretary of a surgical society arranges a "boxing match" between you and your rival, and we who listen to you both are supposed to make a decision on points, a knock-out being too much to hope for. But your supporters, and the other fellow's, each go away muttering: "The two series aren't comparable. I think Tweedledum (or Tweedledee) is right." And there the matter rests, a colossal waste of time, but good fun.

Physicians do these things better, I suppose because they do not invent the drugs they use and because no great technical prowess is required in getting a patient to swallow a pill.

They really get somewhere with their clinical trials. Perhaps we may rise to these heights one day.

And now a few words about conditions today. There is this antinomy between practice and research, inherent in clinical medicine because practice is often unpredictable and sometimes urgent, whereas research requires freedom from interruption. Yet many great men and some not so great have overcome it; it should not be beyond us.

The difficulties can be aggravated by hostility between those interested in practice and those with a taste for clinical investigation. There are faults on both sides. The professorial units, for example, sometimes remain aloof in our medical schools. I recall a remark of one of our greatest professors of medicine who deplored the " insufferable intellectual arrogance " of some heads of academic clinical departments. On the other side, I recall an occasion when an applicant for a senior appointment at one of our greatest postgraduate teaching hospitals was rejected on the advice of its senior surgeon because, we are told, a man who like this candidate had done outstanding research work could not possibly be much good as a surgeon. I knew the man intimately, but my protest was unavailing. As it turned out all was well for he soon obtained an even better appointment in a more enlightened place.

I have known other good junior men, doing excellent research, who were unhappy because they feared that unless they devoted all their energies to clinical practice they would fall behind in the stern competition for senior appointments. Yet not one of them suffered a setback.

The moment our good young investigator attains consultant status his new-found freedom is assailed from two quarters. The temptation to " fill up his sessions " is understandably strong and if he does his work conscientiously—as he will, being a good man—he will hardly have time or the freshness of mind required for continuing his research. And then there are the claims of private work.

I am certain that private consulting practice is the greatest of all hindrances to research. The solution propounded by the Goodenough Committee (following on the Haldane Committee) and increasingly put into effect during the last fifteen years was the establishment of full-time clinical units in all teaching hospitals. There is not the slightest doubt that these units have given scientific medicine a great boost. But this is not the whole answer. The professorial system has difficulties of its own, which I will not trouble you with now; and it is harvesting only a part of the great field. Furthermore, I suspect that a little consulting practice has a healthy disciplinary effect on the clinician. The difficulty is keeping it within strict limits. It can be done by the gentle art of saying NO. This will have to be learnt and applied rigorously by a man who is not content to be the plaything of circumstance, and it is a cardinal prerequisite if time is to be found for research.

It was hoped that the Clinical Research Board would foster enterprise wherever it was found. It was to be a handmaiden of the National Health Service, but it has, in fact, developed as a committee of the Medical Research Council, and has not yet acquired a mind of its own. Nevertheless, this Board is manned entirely by clinicians, and has funds at its disposal for the support of the enterprising part-timer. When I was a member the Board was still skating gingerly round the nub of the problem, how to make provision for the part-timer able and keen to engage in research, so that a certain part of his week be set apart for this purpose. The difficulty was to find a way of decanting him back painlessly and renewing his hospital sessions if he or the Research Board came to the conclusion that his fruitfulness in research was petering out. I think this problem will be solved before long. Money for research expenses is, fortunately, not hard to come by, if a project is a sensible one.

The burden of my song is this. We are primarily practitioners and our work is peculiarly satisfying. But the opportunities that we alone enjoy call for the exercise of scientific method: this requires discipline, much thought and a certain amount of time.

For a number of years past I have been a privileged person; I have not had to wrestle with the mass of routine work that burdens many of you, my friends. Yet I trust I do not underestimate the difficulties of escaping from the servitude of practice. Many of you have shown that it can be done. In this Association there are men who by the careful ordering of their lives and undoubted sacrifice are adding to knowledge, amateurs in the strict and best sense of the word, doing the job for the love of the thing. Their example is worth more than all my precepts.

REFERENCES

ARNOTT, W. W. (1955): The Climate of Discovery. *Lancet*, **ii**, 783.

RAMÓN Y CAJAL, S. (1951): *Precepts and Counsels on Scientific Investigation; Stimulants of the Spirit*. Translated by J. M. Sanchez-Percz. Edited and annotated by Cyril B. Courville. California: Pacific Press Publishing Association, Mountain View.

WALSHE, F. M. R. (1948): Harveian Oration. *The Structure of Medicine and its Place among the Sciences*. Edinburgh: E. & S. Livingstone.

WALSHE, F. M. R. (1950): Linacre Lecture. Humanism, History, and Natural Science in Medicine. *British Medical Journal*, **ii**, 379.

WALSHE, Sir F. (1956): Medicine in the Framework of the University. *British Medical Journal*, **i**, 1,441.

THE ACCIDENT SURGERY OF MOTORWAYS*

PRESTON A. WADE, NEW YORK, UNITED STATES OF AMERICA

I am happy for this opportunity to extend the gratitude of American surgeons to British surgery for its contribution to our development. Two of the periods of greatest progress in surgery of trauma occurred as the result of two world wars. The advances made during these periods were, in large part, due to British surgeons. During the first world war Sir Robert Jones developed the speciality of orthopaedic surgery so that it became a major branch of medicine rather than a minor subdivision of general surgery. During the second world war Sir Reginald Watson-Jones made great contributions in the training of young surgeons in the treatment of injuries. Many of our army surgeons stationed in England during that war were afforded the wonderful opportunity both of reading the works of Sir Reginald and of attending his courses and profiting from his teaching at first hand. These young surgeons returned after the war, greatly inspired, and with tremendous enthusiasm for the wisdom and knowledge thus imparted to them. It is therefore a great personal pleasure for me to have the opportunity to express our gratitude for the tremendous strides our young men have made in the surgery of trauma as a result of his efforts.

Over the past fifty years the relentless pace of our civilisation and the never ending demand for rapid action has made speed in transport a necessity to our society. We seem unwilling, and indeed we are unable, to halt this increase in speed, and we shudder to think of what may be in store for us in the next fifty years. Unfortunately, the inevitable price we pay for speed is injury and death. In spite of all our efforts to the contrary, speed and death will always go hand in hand.

In 1958 in the United States 38,000 people were killed and 1,400,000 injured in consequence of crashes on the highways (National Safety Council 1959). We are, therefore, faced with a very serious public health problem, one in which the medical profession has a great responsibility. As in any public health threat there are two phases to be considered: the first is the problem of prevention, and the second concerns the treatment of the individual involved.

PREVENTION

Speed and failure of judgment on the part of the driver are the most important factors in the causation of motor accidents, and, human behaviour being what it is, it is obvious that even with our greatest efforts only a small percentage of accidents can be avoided. Education in driving, disciplinary laws, enforcement of the laws, and improved road engineering are all important and necessary in the effort to reduce the number of accidents, but these are primarily the responsibility of government agencies, and the medical profession is only indirectly involved. Since we must accept the inevitability of motor crashes as a consequence of rapid travel, one of the measures we may take to prevent injury and death is the protection of the passenger in the motor car. Great advances have been made in the interior design of cars to protect the passenger.

In 1942 De Haven, a research fellow in physiology at Cornell University Medical College, investigated the factors involved in unusual survivals after free falls and attempts at suicide. From his studies he was able to suggest certain changes in the interior design of cockpits of aeroplanes to prevent injury to the pilot in a crash. During the war he continued his research for the Armed Forces, and the improved designs of the cockpits of fighter planes did much to decrease the mortality of pilots in crashes. The natural outcome of this research was to continu

* Condensed from the second Watson-Jones Lecture delivered at the Royal College of Surgeons of England on November 10, 1960.

the same studies in relation to motor cars. With the support of national agencies the Auto-Crash Injury Research Department was established at Cornell University Medical College, and a comprehensive statistical study of many thousands of crashes was made from material gathered in many states (Braunstein 1957). These studies showed that there were certain factors that increased the danger of severe and fatal injury. One of the most important findings had to do with the ejection of the passenger from the motor car at the time of collision (Braunstein, Moore and Wade 1957). In the past it has often been considered fortunate if the occupant was thrown out of the car at the time of collision, because of the mistaken idea that the danger was greater if the passenger remained in the vehicle. But research indicated that the risk of serious or fatal injury was five times greater if the occupant was ejected from the vehicle (Table I). The relative safety of remaining in the vehicle is well known to stunt drivers, who have no fear of roll-over crashes if they are strapped to a solidly fixed seat and the doors are wired tight to prevent opening.

TABLE I

RISK OF INJURY AS RELATED TO EJECTION OR NON-EJECTION

	Percentage of occupants with:			
	Moderate/fatal grade injury	Severe/fatal grade injury	Dangerous/fatal grade injury	Fatal grade injury
Not ejected . .	23·9	9·9	5·5	2·6
Ejected . . .	49·9	36·4	26·6	13·3
Injury ratio Ejected: not ejected	2·1:1	3·7:1	4·8:1	5·1:1

Certain parts of the interior of the motor car were found to be more dangerous than others during a crash. The steering assembly, windscreen and instrument panel were often the cause of injury. The fact that the front seat often tore loose, throwing the passenger forward against the dashboard, was found to be another cause (Table II).

On these findings, the Auto-Crash Injury Research Department made certain suggestions concerning the interior design of motor cars. They recommended: 1) provision of a safety belt for all occupants; 2) padding of the instrument panel and removal of protuberant knobs and keys; 3) change in design of the steering assembly, the wheel being dish-shaped to catch the body of the driver and prevent the impaling action of the steering rod; 4) improvement of door locks to prevent doors from opening on impact; and 5) firm fixation of the front seat to the chassis. These suggestions impressed the Trauma Committee of the American College of Surgeons, and on its recommendation the Board of Regents of the College transmitted them to the five major motor manufacturers in America with the request that these changes be made. Most of the manufacturers complied, and one company incorporated all of these suggestions in its 1956 model. Some of the improvements, such as padding of the dashboard and provision of seat belts, were offered only as optional features at added cost to the buyer. This was unfortunate because most of the public was not aware of the value of the safety features. One of the manufacturers centred its advertising campaign on these safety features, but unfortunately its sales figures showed a severe drop, and the experts attributed this decline to the accent on safety. Apparently safety suggestions are not popular with the man buying a car. Nevertheless the manufacturers are continuing to include many of the safety features in their new models.

Since these changes in motor car design were made in 1956 the Cornell group has investigated the effectiveness of safety features in some 2,750 accidents producing injury in rural areas. The findings indicated a highly significant decrease in the injury potential of the

motor car. Table III shows a comparison on the basis of incidence of door openings, of ejection and of the risk of dangerous or fatal injury. The greatest improvement in risk of ejection was 48·7 per cent and the greatest decrease in risk of dangerous or fatal injury was 29 per cent. Both of these changes are significant at the 95 per cent level of confidence.

TABLE II

MAJOR CAUSES OF INJURY

	Percentage of occupants injured to:			Order of importance*
	Any degree	Moderate/fatal degree	Dangerous/fatal degree	
Steering assembly	29·4	8·4	2·5	1
Ejection	14·6	6·9	3·2	2
Instrument panel	20·6	4·2	·7	3
Windscreen	16·9	4·6	·6	4
Backrest of front seat (top portion)	11·0	2·4	1·1	5
Door structures	7·7	2·4	·5	6
Backrest of front seat (lower portion)	15·1	2·5	0	7
Front corner post	2·0	1·2	·7	8
Flying glass	3·0	·5	·02	9
Top structures	1·2	·6	·2	10
Rear view mirror	2·2	·6	·02	11

* Based on 1) the number of occupants actually exposed to the injury-hazard of the object, 2) the frequency of injury caused by the object, 3) the degree of injury caused by the object.

TABLE III

SAFETY OF 1956 AND PRE-1956 CARS

(Based on 2,750 rural accidents)

Year of manufacture	Percentage risk of door opening in:			Risk of ejection:	Risk of dangerous/fatal grade injury:
	Overturning accidents	Accidents without overturning	All types accidents	Percentage of occupants ejected	Percentage of occupants with dangerous/fatal grade injury
1940–1955	75·1	38·1	46·3	11·9	9·3
1956	60·0	25·3	34·0	6·1	6·6
Difference in risk of front doors opening	15·1	12·8	12·3	5·8	2·7
Maximum improvement demonstrated	20·1	33·6	26·6	48·7	29·0

In another investigation of the effectiveness of seat belts, 300 cars equipped with such devices, involved in crashes, were studied (Braunstein *et al.*, *loc. cit.*). By reviewing 4,000 control cases it was possible to match closely eighty-one cars with seat belts and eighty-one without. The control and experimental groups were identical as to type of accident, area of principal impact, direction of principal force, speed at impact, frequency of door opening, number of cars per accident, make of car, weight of car, year of manufacture, and seated area of occupants.

TABLE IV

EFFECT OF SAFETY BELTS

(As demonstrated by a study of 236 occupants of 162 cars in rural accidents)

Type of injury	Frequency of injury (*per cent*)		Maximum improvement (*per cent*)
	No safety belt	Safety belt	
All degrees	75·5	29·9	60·4
Moderate/fatal	23·0	9·2	60·0
Dangerous/fatal	3·6	1·0	None (probably due to small sample-size)

Both groups were identical as to: 1) type of accident, 2) area of principal impact, 3) direction of principal force, 4) speed at impact, 5) frequency of doors opening, 6) number of cars per accident, 7) make of case car, 8) weight of case car, 9) year of manufacture of case car, 10) seated area of occupants.

The results of the comparison are listed in Table IV. Some injury was sustained in 75 per cent of persons with no belt, but in only 29·9 per cent of those with seat belts. Moderate injuries were reported for 23 per cent of the group with no belts and 9·2 per cent of those with belts. Fatal injuries were reported in 3·6 per cent of those without belts and in only 1 per cent of those with belts. All degrees of injury were decreased to a degree which was statistically significant at a very high level of confidence. Three examples showing the increased safety provided by seat belts will be quoted from the eighty-one paired accidents studied. In the first pair of cases (Cases 1 and 2) each car was involved in a head-on collision at the same speed, causing similar car damage (Figs. 1 and 2). The driver without the seat belt was killed, but the other survived with minor injuries. In the second example (Cases 3 and 4) each of the accidents involved a right front corner impact with another car at thirty-five to forty miles an hour. The damage to the car was similar in each case (Figs. 3 and 4). The driver wearing the seat belt was uninjured, but the other driver was thrown violently against the right front door and sustained multiple injuries, including facial contusions, fractured clavicle, fractured ribs, punctured lung with haemo-pneumothorax, and fractured patella. In the third example (Cases 5 and 6) both accidents occurred at seventy miles an hour and the damage to the two cars was similar (Figs. 5 and 6). The driver wearing the seat belt sustained minor contusions, whereas the other driver was partly ejected through the windscreen and sustained severe injuries which included multiple facial lacerations with avulsion of the right ear, fracture of the right femur, and multiple lacerations about the right knee.

Another remarkable survival of a driver wearing a seat belt is illustrated in Figure 7. In this accident the car was completely sheared in two by a tree, and the front half of the car was thrown fifty feet across a field. The driver, who was wearing a seat belt, walked away from the accident unhurt.

It cannot be expected that a seat belt or other safety device will afford protection in accidents that occur at excessive speeds (Fig. 8). The safety factors that may be incorporated by the motor manufacturer will continue to be helpful in saving lives, but those that depend on the judgment of the individual driver and passenger will be difficult to apply. It is the duty of the medical profession to help in the effort to educate the public in this respect. The American Medical Association, the United States Public Health Service, and the National Safety Council are cooperating in an effort to expand the use of seat belts. Much progress has been made, particularly in the acceptance of the seat belt by large companies controlling big fleets of cars and trucks. It is hoped that in the future all cars will be equipped with seat belts and that the public will be educated to use them.

TREATMENT

The major responsibility of the medical profession to the accident victim lies in the treatment of his injuries. In the opinion of the Trauma Committee of the American College of Surgeons the care of the injured patient in the United States is not all that it should be, and we are now investigating the present state of the ambulance services and of emergency treatment in hospitals in America.

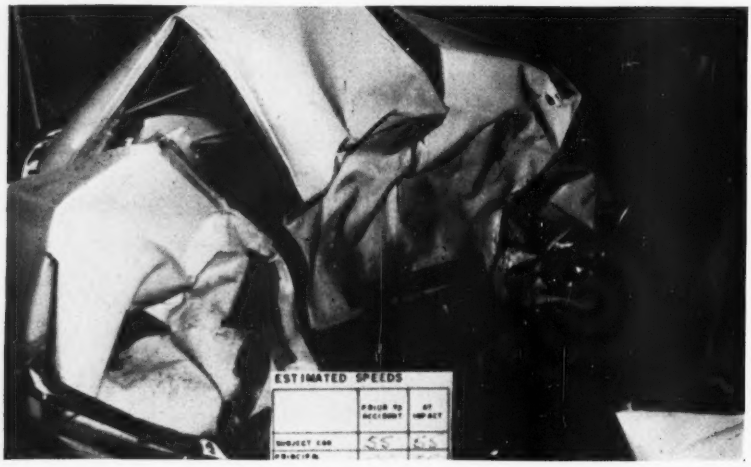


FIG. 1
Case 1—State of car after head-on collision. The driver was not wearing a seat belt and was killed.

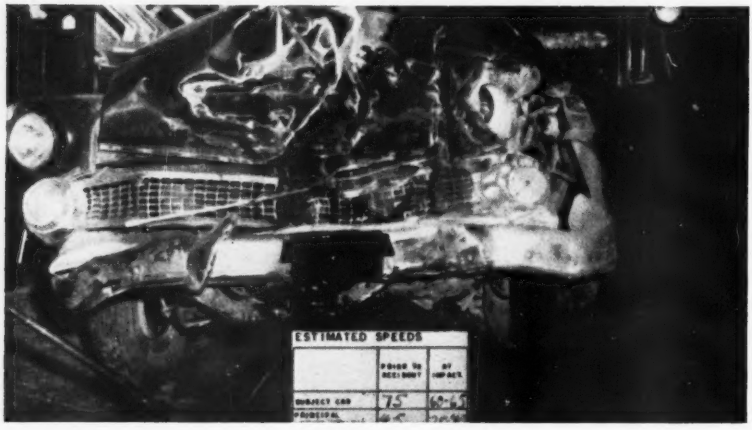


FIG. 2
Case 2—Car similarly damaged. The driver, who was wearing a seat belt, sustained only minor injuries.

The transport of the injured may be a major factor in the outcome of the treatment of his injuries. In the United States there are few cities and rural communities in which an injured person is transported under ideal conditions. Through its Trauma Committee, the American College of Surgeons is continuing, as one of its major efforts, an attempt to improve this situation.

Since the excitement and hysteria aroused by an accident usually stimulate all concerned to use as much speed as possible in their actions, the ambulances are usually driven at excessive speed on the highways, even when not answering an emergency call. The thrill of speed and the feeling of special privilege are too great a temptation for the average ambulance driver to resist. In a study of 2,500 cases of patients taken to hospitals in Flint, Michigan, it was found by Curry and Lyttle (1958) that undue haste was not essential to the patient's welfare.

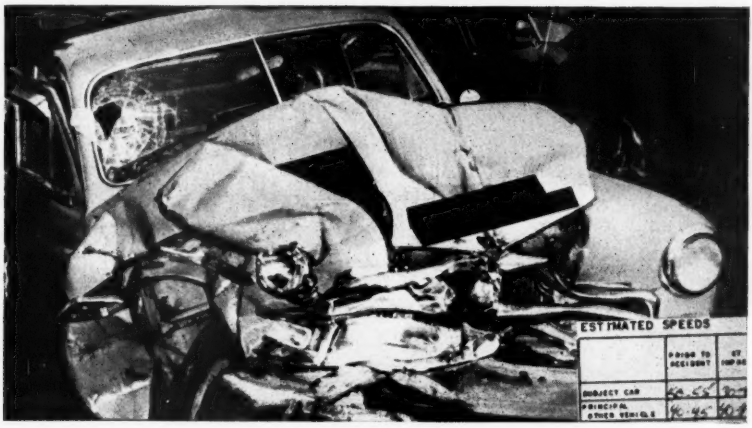


FIG. 3
Case 3—State of car after front-corner impact. The driver was not wearing a seat belt and sustained multiple injuries.

FIG. 4
Case 4—Car similarly damaged. The driver, who was wearing a seat belt, was uninjured.

In their opinion in only one case would moderate delay in transport have caused a fatality. In only forty-five cases was the interval between the accident and the patient's arrival at hospital considered to be significant in determining the patient's course. In nine cases, on the other hand, a rough, weaving ride to the hospital may have produced death or permanent disability. In my opinion the speeding ambulance kills more people than it saves. Until there comes a time when ambulances are manned by responsible and qualified drivers who will speed only when it is necessary, and who will use careful judgment in tempering regulations,

ambulances and other vehicles carrying the injured should be required to obey the usual traffic laws.

The quality of care received by an injured patient in the emergency room is also now under scrutiny by a special group under the auspices of the Trauma Committee of the American College of Surgeons (1960). We believe that emergency room care can be improved and we hope that this investigation will result in the establishment of minimal standards which hospitals will be encouraged to meet.

FIG. 5
Case 5—State of car after high-speed impact. The driver, not wearing a seat belt, was severely injured.

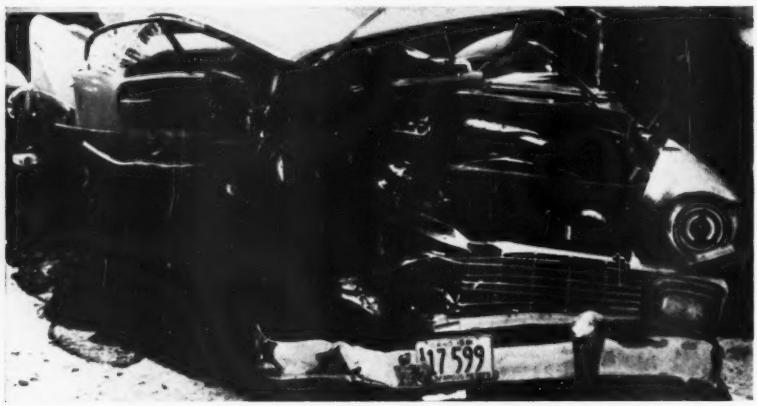


FIG. 6
Case 6—Another car similarly damaged. The driver was wearing a seat belt and sustained only minor contusions.

The injured workman in large industrial centres in America receives excellent care because his accident occurs during working hours and he is taken immediately to a well equipped hospital staffed by surgeons experienced in the type of injury which may be expected in the industry in question. A motor accident, in contrast, often occurs in an out-of-the-way place at an unpredictable hour. The victim is usually taken to the nearest hospital, which may not be equipped to handle an emergency.

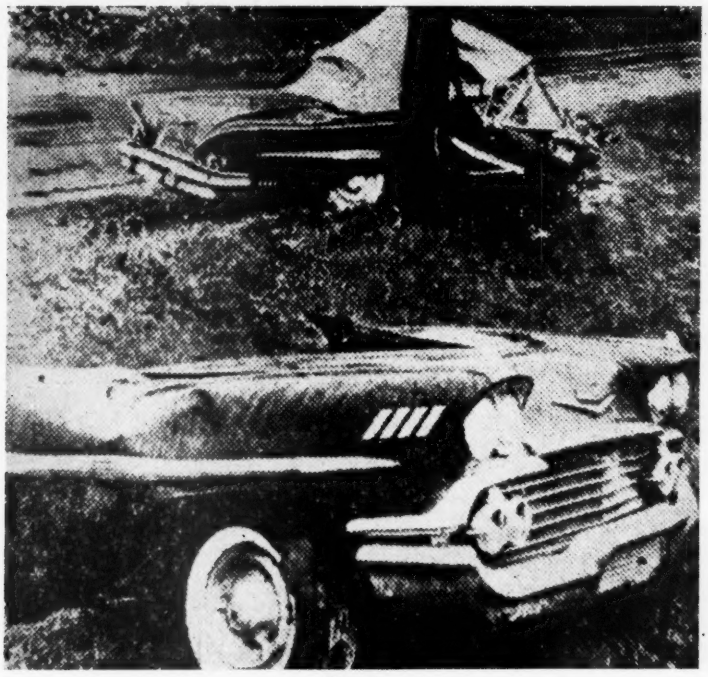


FIG. 7

This car was cut in two by the tree with which it collided. The driver was wearing a seat belt. He walked away uninjured.

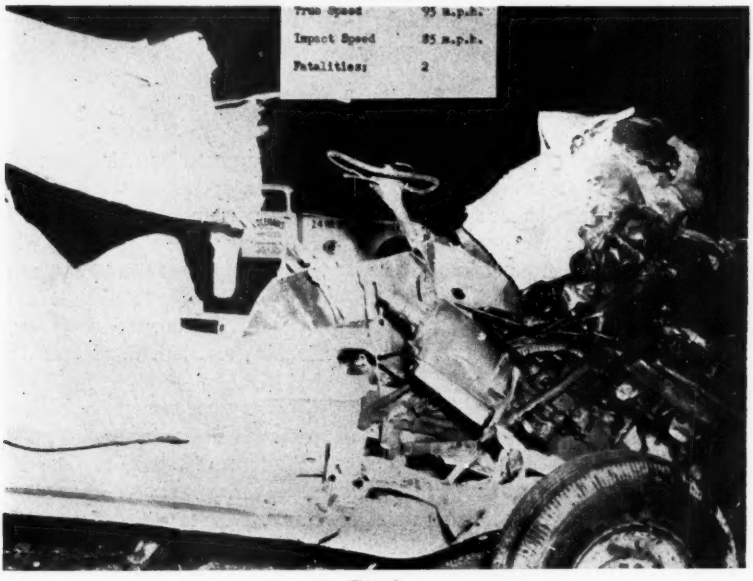


FIG. 8

Even with the protection of seat belts it cannot be hoped that severe injury will be prevented in high-speed accidents. The two occupants of this car were both killed instantly despite seat belts.

The following cases illustrate the multiple nature of the injuries so frequently seen in hospital emergency rooms. Each presents serious problems of treatment in many different fields of surgery.

Case 7—Figure 9 shows the wreck of a car in which two passengers, neither wearing a seat belt, were severely injured in a head-on collision. The passenger on the left front seat sustained a comminuted fracture of the right femur, a ruptured spleen, a severe head injury, fractured ribs and division of an extensor tendon. He was unconscious and shocked on admission. After assessment of the head injury a laparotomy was performed within two hours and the spleen was removed: the femoral fracture was treated by Russell traction. The laceration of the extensor tendon of the left ring finger was not recognised for eight days (much to our chagrin) and only when the patient complained that he could not extend the finger. How often must we tell ourselves and our assistants that especially in the presence of a serious injury we must make a thorough examination to discover even a relatively minor injury? Division of the extensor tendon of the ring finger is not a minor injury to this photographer.

FIG. 9
The car in Case 7.

The driver of the car sustained a severe head injury, a posterior dislocation of the right hip, with fracture of the posterior lip of the acetabulum, an open fracture of the patella, and multiple fractures of the ribs on the right side with dangerous haemo-pneumothorax. Treatment was by immediate needle aspiration of the chest, followed by thoracotomy, and within a few days operative correction of the dislocation of the hip and repair of the extensor mechanism of the knee. A study of multiple injuries at the New York Hospital (Wade 1952) suggested that when a posterior dislocation of the hip is associated with injury of the femur, knee, or tibia on the same side the hip injury may be overlooked, and it is important that this be considered in every case of multiple injury.

Case 8—The second illustrative case is that of a pedestrian struck by a car on the streets of New York. An investigation of 100 serious and fatal pedestrian injuries being carried out by the Cornell University Trauma Research Department (Wade, Braunstein, McCarroll, Cooper, Seremetis and Weinberg 1961) has shown that most of these serious and fatal accidents involve an elderly pedestrian, often under the influence of alcohol, attempting to cross a street against traffic lights and at a place where crossing is illegal. Such was the case with this man, and the injuries he sustained were typical of many in the series. He sustained multiple fractures of the right transverse processes of the lumbar vertebrae, fractures of the right eleventh and twelfth ribs and fractures of the right ilium and of the sacrum. He also sustained a dislocation of the left

knee, and fracture of the right tibia and fibula. He suffered concussion of the brain and multiple contusions and abrasions. Here was a patient who needed immediate treatment for the dislocated knee with its circulatory embarrassment and nerve damage, and treatment for shock and for the brain injury, as well as careful observation of possible kidney and intra-abdominal injury. He developed a massive retroperitoneal haemorrhage (one of the most common unrecognised causes of death in our pedestrian series) with a large visible and palpable mass in the right flank. But more important than any of these, he exhibited the classical signs of the crush syndrome with kidney damage and anuria. Figure 10 illustrates the pattern of kidney function

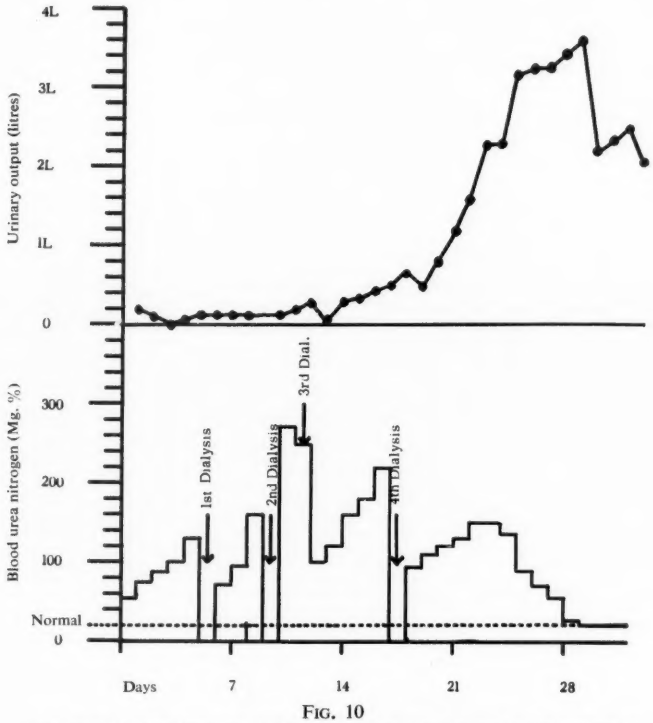


FIG. 10

Pattern of kidney function in the four weeks after injury in the patient in Case 8. After four dialyses on the artificial kidney the kidney parenchyma began to function.

in the four weeks after the accident, during which the kidney parenchyma began to function again after eighteen days, after four dialyses on the artificial kidney. Surely this patient needed immediate care for his many injuries, but also the wise observation of the surgeon in charge, who could recognise the onset of the anuric symptoms and institute proper treatment.

Case 9—The final example is of the type of case that may be seen in any hospital emergency room at any time, and in which the patient will need expert care if life and limb are to be saved. Figure 11 shows the damaged vehicle in which a man and wife were riding at speed when they struck a concrete pillar on a highway. Neither was wearing a seat belt, and the driver was killed instantly, impaled on the steering column. His wife was thrown violently forward and then ejected to the roadway. A passing taxi cab brought her to the hospital, unconscious, in great respiratory distress, pulseless, and in severe shock. Her injuries included fracture of the skull, fracture of the jaw, segmental crush injury to the chest with fractures of six ribs on either side, and with paradoxical respiration, a transverse fracture of the body

of the eighth thoracic vertebra, and fracture of the pelvis. She had sustained injuries to the abdomen and there was blood in the catheter specimen of urine. She had sustained a severe crushing injury to the right lower limb with avulsion of skin, and a severe open fracture of both bones of the left leg with extrusion of the fragments through the wound.

It was necessary to treat her for shock immediately and to maintain respiratory exchange. After urgent blood transfusion and tracheostomy the wounds were debrided, traction was applied to the chest wall, the fracture of the left leg was put in skeletal traction and the fractured mandible was wired. After a slow convalescence she eventually made a very good recovery.

FIG. 11
The car in Case 9.

The follower of the school of pure fragmentation in surgery might say that, ideally, this patient needed a " specialist " in neurosurgery, in maxillo-facial surgery, in orthopaedics, in abdominal surgery, in thoracic surgery, in urological surgery and in plastic surgery as well as the services of a radiologist, a laboratory expert, an anaesthetist, and experienced nurses.

It is unrealistic to hope that all seven of those surgeons could be present at the reception of any patient, and, indeed, such a cluster of experts would be confusing and confounding. One competent surgeon with a broad knowledge of trauma in every field is quite capable of caring for the early therapeutic needs. In directing total care he would use the services of consultants in each field at the appropriate time and in reasonable sequence when the need arose.

In studying the adequacy of emergency room care in American hospitals we should like answers to the following questions involved in coordinating the care of a patient similar to the one we have used as an illustration. Would the patient be seen immediately by someone competent to judge her needs? Would her respiratory embarrassment be corrected by proper treatment? Would a qualified surgeon be present to do a tracheostomy when necessary? Would a qualified observer assess the head injury and decide whether or not surgery was indicated? Would the abdominal injury and bladder injury be promptly recognised? Would laboratory, blood bank and radiological facilities be available in the emergency room? Would the wounds and open fracture be properly debrided and would these operative procedures be undertaken promptly with due consideration of the other injuries? And, above all, would

one responsible surgeon see the patient immediately after admission and thereafter direct her care throughout her entire illness?

We believe that in many hospitals this patient would receive excellent care, but fear that in a far greater number these requirements would not be met.

This problem is a fundamental one of education as well as organisation. In our medical schools today the vast amount of information that the overworked medical student is expected to absorb takes so much time that each departmental head feels that his discipline is underrated in the curriculum. Nevertheless, if we are to expect that the patient with multiple injuries will be cared for by a qualified surgeon, we must prepare to educate the surgeon and give him an opportunity to develop in the many and varied aspects of trauma therapy. The teaching of trauma in the medical schools deserves much greater consideration than it is now given, not so much in extra time as a separate speciality, but in increased emphasis on trauma in every field, so that the medical student can see all of the facets of trauma during his undergraduate education. Postgraduate training in trauma needs greater emphasis in the programmes both of general surgery and of the specialities. In recent years this has been recognised by the American Board of Surgery, which now not only requires training in trauma, but also includes the subject as a much more significant part of the written and oral examination than it did in former years. Some training programmes in general surgery do not include the treatment of fractures or other injuries. This is indeed a great mistake, because no young man who has not been trained in trauma can be considered a fully trained surgeon. In many orthopaedic training programmes the emphasis is on the purely orthopaedic problems, and many of the trainees have little experience in the treatment of multiple injuries.

These deficiencies are well known, and steps are being taken to correct them. The organisation of trauma treatment has received increasing consideration in the past few years and much good may result from this. The American Board of Surgery once considered establishing a separate speciality for surgeons interested in trauma, and several years ago an effort was made to give an examination qualifying in the surgery of trauma certain already certified general surgeons. This move was strongly opposed by orthopaedic surgeons because they feared that the so-called trauma surgeons would then absorb all fracture cases. Furthermore, the argument has commonly been advanced, by sources from both general surgery and orthopaedic surgery, that it is impossible for any surgeon to become competent in the treatment of trauma in every field. The obvious implication is that each surgeon must confine himself to the treatment in his own speciality, and should not treat conditions in other fields. As we have already seen in the cases discussed here, such anatomical fragmentation of responsibility is not to the advantage of the patient. Although it is quite correct that no one can learn all there is to know about every speciality, he can do the patient the greatest service if he knows much about the care of injuries sustained in those areas other than his own field, and if he knows a great deal about the emergency care of every body area.

Much of this disagreement is the result of professional jealousy and fear of financial loss. This may not be true in Britain, but it is decidedly so in my country. The general surgeon has resisted the efforts of orthopaedic surgeons to enter general surgical fields, and the inclusion of hand surgery in orthopaedic training is often resented by the plastic surgeon as well as the general surgeon. The neurosurgeon feels that the orthopaedic surgeon should not attempt to repair nerves or operate upon intervertebral discs. The vascular surgeon resents the fact that orthopaedic surgeons may learn to perform vascular grafts in the presence of other injuries to the limb. What utter nonsense. What difference does it make to the injured crash victim whether or not his surgeon has a certificate in a certain field of surgery: he demands and deserves the best treatment he can get from a surgeon trained in the care of trauma.

It would be ideal if every injured patient could be taken to a specialised hospital or to a special unit of a general hospital where these experienced surgeons, with adequate help, equipment and supplies could remove a ruptured spleen, operate upon a middle meningeal

haemorrhage, debride and reduce an open fracture, treat a stove-in chest, and still have access to consultation by other specialists. The regional organisation of medical trauma centres is a worthy idea and may be the answer to the problem, particularly in communities where distances are not great and where population is concentrated. However, in certain regions in the United States, where distances are great, hospitals are sparsely distributed, and qualified personnel necessarily few in number, such a concept is impracticable. In Austria and in Hungary, where accident hospitals are the rule, the experiment has been eminently successful, as has your experience in Britain, and ours too, in a few centres; but in the United States we feel that it is still our duty to emphasise the need for better care in every hospital emergency room. Later, perhaps, there may be established more regional units to which organised ambulance services may transport the injured patient.

Orthopaedic surgery now has a remarkable opportunity to become the trauma speciality of the future. If the orthopaedists do not meet this challenge then it must be the general surgeon who must develop the traumatologist, a designation hated by both orthopaedic and general surgeons. There is no reason why the general surgeon cannot be trained to treat all injuries to bones and joints, and there is no reason why the orthopaedic surgeon who has a basic surgical training cannot be trained to treat injuries in other fields so that he can handle chest injuries, abdominal injuries and vascular injuries. He may not be adept at hypophysectomy and removal of brain tumours, but he most certainly could learn to take care of middle meningeal haemorrhage. He may not have time to learn the intricacies of lung cancer, but he can certainly learn how to handle a stove-in chest and pneumothorax. He may not be proficient in cardiac surgery, but he must learn the technique of vascular repair and blood vessel grafting. If orthopaedic surgery takes up the challenge and trains its young surgeons in general surgery before he takes on his training in orthopaedics, and if the directors of surgery have enough insight into the problem to allow him to continue his work in fields other than that of bones and joints, the orthopaedist will become the traumatologist of the future. One of the world's most renowned orthopaedic surgeons told me not long ago, facetiously perhaps, but with certain seriousness: " You should remember always that the orthopaedic surgeon is the general surgeon of the future, whereas the general surgeon of the past has become more and more of a specialist."

In any case, whether the trauma expert be primarily a general surgeon or an orthopaedic surgeon we may expect that those injured in road accidents will in future receive expert care by trained surgeons, in organised accident units of general hospitals or specialised accident centres. And in the care of these broken bodies may we always consider not so much in our belief as in our practice, and still more in our teaching, the inspiring admonition of the great man for whom today we are met to show our love and respect: " The patient must understand his disability; he must again gain confidence and be inspired; his doubts must not become anxieties; his fears and misgivings must be dispelled; his social problems must be solved; he must be reassured; he must not fear the future."

REFERENCES

BRAUNSTEIN, P. W. (1957): Medical Aspects of Automotive Crash Injury Research. *Journal of the American Medical Association*, **163**, 249.

WADE, P. A., BRAUNSTEIN, P. W., MCCARROLL, J. R., COOPER, W., SEREMETIS, M., and WEINBERG, S. (1961): Fatal Pedestrian Automotive Accidents. *In Press.*

BRAUNSTEIN, P. W., MOORE, J. O., and WADE, P. A. (1957): Preliminary Findings of the Effect of Automotive Safety Design on Injury Patterns. *Surgery, Gynecology and Obstetrics*, **105**, 257.

CURRY, G. J., and LYTTLE, S. N. (1958): The Speeding Ambulance. *American Journal of Surgery*, **95**, 507.

DE HAVEN, H. (1942): Mechanical Analysis of Survival in Falls from Heights of Fifty to One Hundred and Fifty Feet. *War Medicine*, **2**, 586.

NATIONAL SAFETY COUNCIL (1959): *Accident Facts.* Chicago, Illinois: National Safety Council.

REPORT OF COMMITTEE ON TRAUMA (1960): *American College of Surgeons.*

WADE, P. A. (1952): The Care of Multiple Injuries. *Surgical Clinics of North America*, **32**, 667.

LOW-ANGLE FIXATION IN FRACTURES OF THE FEMORAL NECK

R. S. GARDEN, PRESTON, ENGLAND

From the Orthopaedic Department, Preston Royal Infirmary

Fracture of the neck of the femur continues to be regarded as "the unsolved fracture," but its claim to this distinction becomes increasingly insecure. Since the introduction of the Smith-Petersen nail in 1931, unrelenting endeavours have been made to solve this problem, and the literature reveals a wide variety of anatomical studies, statistical reviews, new methods of reduction, fixation and bone-grafting techniques. The overall picture is one of some confusion but two elementary points of universal agreement are seen to emerge: reduction must be perfect; and fixation must be secure.

No one will deny the advantage of perfect reduction in the treatment of this or any other fracture, but accurate reduction is not essential to union in fractures elsewhere. Nevertheless, it is generally agreed that in fractures of the femoral neck full reduction is of particular importance. In regard to fixation many new methods of treatment have been devised. Wires, nails, multiple screws and lag bolts, combined bone-grafting procedures, sliding and compression devices have all been described, each striving to achieve rigid apposition of the fracture surfaces. As these new appliances and techniques have been introduced, there has been an increasing tendency to rely on the safeguard of shaft fixation or to use a more vertically placed fixation device.

Low-angle nailing was advocated in this country by Brittain in 1942, by Burns and Young in 1944 and since then by many other surgeons. Dickson (1953) stated that "the Jewett-type nail placed in low position so that it practically rests on the calcar femorale . . . apparently increases the stability, and thus increases the likelihood of bone healing at the fracture site." Küntscher (1953) believed that the low-angle nail neutralised the muscular forces acting upon the fracture and, since the nail entered the head obliquely, prevented rotation because the head was pressed hard against the fracture site in the direction of the nail. He maintained that low-angle nailing produced a fixation eight times stronger than that afforded by the Smith-Petersen operation. Lastly, he stated that after this method of fixation the full weight of the body could be supported. These are formidable claims.

The present inquiry was prompted by the assertion that low-angle nailing could sustain the body weight. It was felt that confirmation of this claim would at the same time confirm a singular advance in the management of femoral neck fractures, which are largely confined to an age group in which degenerative processes, already far advanced, can easily be accelerated by prolonged recumbency.

INTERNAL FIXATION

Internal fixation, whatever its form, must in some way respect the architecture of the internal weight-bearing system in the proximal end of the femur if both weight bearing and fixation of the fracture are to be achieved at one and the same time. It is suggested that the lamellae of the internal weight-bearing system are a spiral continuation of the circumferential lamellae in the femoral shaft, and the forces acting upon the proximal end of the femur are primarily compressive and secondarily torsional in nature (Garden 1961). The medial lamellae in the internal weight-bearing system are directed upwards at an angle of only 3 to 8 degrees with the perpendicular and their rich endosteal lining forms the principal source of bone repair after fractures of the femoral neck (Farkas, Wilson and Hayner 1948). Thus, internal fixation need try to emulate only compressive and torsional elements in the proximal end of

the femur, and its purpose should be to maintain the fragments of the medial arrangement of lamellae in close approximation until healing is complete.

The question then arises whether any form of internal fixation can be expected to maintain the fragments in apposition whilst sustaining body weight as well. Any metallic appliance inserted almost horizontally across the fracture site traverses the thin cortical shell just below the greater trochanter, and then lies in a cancellous bed within the trochanter and neck (Fig. 1). It is difficult to obtain a secure hold on the distal fragment in this way, and the fixation appliance is bound to cut out when weight bearing is undertaken. On the other hand, in oblique or transverse fractures of the femoral shaft early walking may be safe after the insertion of a rigidly positioned intramedullary nail. Body weight is then directed through the fracture

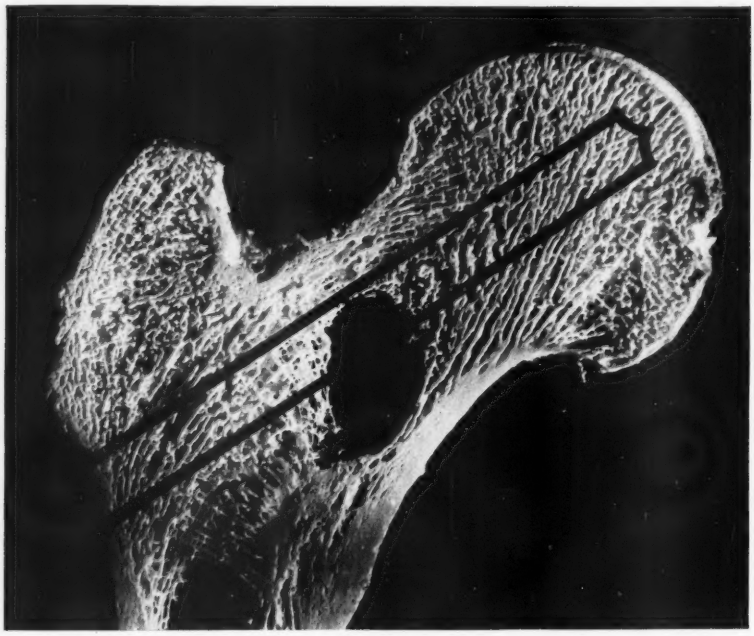


FIG. 1

Frontal section of proximal end of the femur. Position of Smith-Petersen nail to show the insecurity of its fixation in the distal fragment of a subcapital fracture where it pierces the thin lateral cortex just below the greater trochanter, and where it lies in the soft medulla of the neck.

fragments which the nail preserves in normal alignment whilst avoiding other than minimal local strain. But the relatively simple mechanics of the femoral shaft do not apply to its trochanteric area or neck. Here the problem is complicated, indeed created, by the angle between the neck and shaft. Anatomically, this angle is in the region of 127 degrees, but Nature does not recognise the whole of this angle in the transmission of weight-bearing stresses. Backman (1957) considered that the direction of load in the proximal end of the femur coincides with the principal direction of the medial trabecular stream in the internal weight-bearing system, which lies at an angle of no more than 3 to 8 degrees with the perpendicular. This is probably true for the anatomical specimen subjected to compression tests in the laboratory, but, in the living femur which has to contend with both body weight and muscular forces, a slightly less vertical direction should, theoretically, be more exact. The analogy of the intramedullary nail in femoral shaft fractures is thus more applicable than at first appears.

By this analogy, weight bearing in cervical fractures of the femur should be possible by the use of a fixation appliance lying in a near-vertical position within the spiral of the neck, and depending for its purchase upon compact rather than cancellous bone. By directing the body weight through the structure best suited to sustain it, namely the actual bone of the femoral head and neck, an appliance so placed should then maintain the fragments in alignment whilst, at the same time, itself evading the direct strains of weight bearing.

The Smith-Petersen nail was designed to prevent rotation of the fragments in the long axis of the neck, but such rotation is unlikely to occur, because the serrated fragments are firmly apposed and the capital fragment is mobile. The deforming rotation in subcapital fracture takes place in the more vertical mechanical axis of the femur as the affected limb tends to roll outwards. The fragments at first rotate in opposite directions like two cog-wheels in mesh, but as the deformity increases the cogs are disengaged or broken, as it were, and the cervical fragment comes to lie in front of the head. It is with this lateral rotation deformity that internal fixation must contend, and it is this force which causes displacement of a horizontally positioned nail lying in the soft cancellous bone of the distal fragment.

Some protection from this tendency to slip is afforded by shaft fixation with a combined nail and plate. This combination, however, continues to work at a distinct mechanical disadvantage because support is provided at the wrong end of the nail. Lateral rotation forces acting on the point of the nail are transferred to its base, and breaking, bending or twisting at the junction of nail and plate is not unusual.

FIG. 2

Subcapital fracture of the femur treated by low-angle Küntscher nail showing rigid fixation of the nail in the distal fragment where it pierces the strong lateral cortex well below the greater trochanter, and where it lies against the calcar femorale and inferior buttress of the neck.

The unsatisfactory results of the Smith-Petersen procedure are well illustrated by the reluctance of many surgeons to depend upon the trifin nail alone. Some have advocated a combined bone-grafting procedure; some have insisted upon reduction in the deceptive valgus position; some have advised an intertrochanteric or wedge osteotomy; and some—in despair—have resorted to removal of the head and replacement by a prosthesis. King (1939), Patrick (1949) and others have combined a bone graft with the Smith-Petersen pin, but it is noteworthy that their published radiographs show the pin in low position to accommodate the graft above it. Godoy Moreira and Camargo (1957) have also had recourse to the use of a bone graft which, likewise, has displaced their stud-bolt downwards to a position of stability on the calcar femorale. Most workers in this field who have improved their results have done so by ensuring that the two fragments are held in stable contact whilst at the same time minimising the unnatural stresses of metal impinging upon bone.

It is now freely recognised that a nail or screw placed almost horizontally is mechanically unsound, and that any form of appliance for internal fixation must be placed more vertically. The low-angle nail which pierces the strong lateral femoral cortex well below the greater trochanter and which then lies against the buttress of the calcar femorale obtains a rigid two-point fixation in the distal fragment (Fig. 2). One method at least, therefore, can provide an unyielding purchase in this fragment. Unfortunately, it is difficult to establish a similar

hold on the proximal fragment. This is especially true in the subcapital fracture of the aged where the femoral head is so degenerate and friable that internal fixation by any means at present known is, at best, precarious.

SUBCAPITAL SEPARATION

High cervical fractures often follow the most trivial injury, and sometimes there is no injury at all. It is often impossible to determine whether the fracture was the result or the cause of

FIG. 3

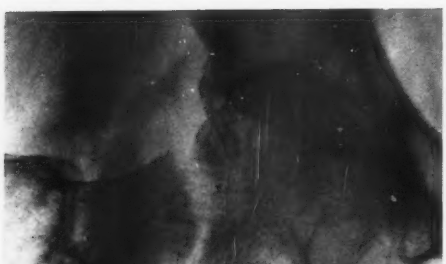


FIG. 4

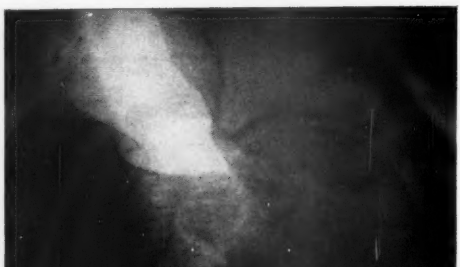


FIG. 5

FIGS. 3 TO 5

Serial radiographs to illustrate the successive stages in the development of a stress fracture of the femoral neck. Figure 3—Initial radiograph. Figure 4—One month later. Figure 5—Eight days later.

a fall, and the resemblance of these injuries to pathological fractures is not easy to ignore. Subcapital separation has been ascribed to degenerative changes in the blood vessels of the marrow (Farkas *et al.* 1948). If this subcapital separation is but the culmination of a natural degenerative process and not the result of an injury, then the use of the term " fracture " in

describing this condition would be misleading. The following considerations suggest, however, that subcapital separation is not entirely a pathological process.

With advancing age the muscles that control the hip joint gradually lose their tone, and therefore their ability to continue the mechanically difficult part which they play in the act of walking. Balancing on one leg becomes progressively more uncertain, and this uncertainty is increased by a decline in the acuity of the special senses. Consequently, the swinging gait of youth and middle age gives way to the shuffling gait of the aged. In response to this redistribution of the weight-bearing stresses, the head and neck of the femur presumably undergo functional realignment of their weight-bearing systems. During the preliminary

FIG. 6

FIG. 7

Figure 6—STAGE I: INCOMPLETE SUBCAPITAL FRACTURE. The " abducted " or " impacted " injury in which the fracture of the inferior cortical buttress is greenstick in nature, and a minimal degree of lateral rotation of the distal upon the proximal fragment creates the radiological illusion of impaction. The medial lamellae of the internal weight-bearing system in the distal fragment lie in abduction as compared with those in the capital fragment which, itself, is adducted. If unprotected, this fracture may at any time become complete.
Figure 7—STAGE II: COMPLETE SUBCAPITAL FRACTURE WITHOUT DISPLACEMENT. The inferior cortical buttress has been broken, but no tilting of the capital fragment has taken place. As in the Stage I fracture, the closely opposed fragments in this complete fracture may succumb to lateral rotation forces and show the classical displacement of " subcapital separation."

process of softening which this must entail, a stress fracture may readily occur. This can sometimes be observed radiologically as it takes place (Figs. 3 to 5), and it may well be that many such incomplete fractures remain unrecognised and heal spontaneously.

The line of cleavage in this stress fracture starts at the upper cervico-capital junction and gradually runs downwards until it meets the inferior cortical buttress of the neck. The strains of weight bearing then devolve largely upon this buttress, which must itself share in the remodelling process. So far this process has resulted in a gradual dissolution of continuity in the internal weight-bearing system, but the final stage entails an actual fracture of the weakened inferior cortex. A minor twist, an unguarded action, an uneven step or, it may be, a normal step, shatters this cortex and subcapital separation is complete. The proximal fragment is then a cone-shaped segment with radiographic evidence of recent fracture showing

mainly at the tip of the cone where comminution is almost always to be found (Fig. 8). Occasionally a greenstick fracture occurs and the so-called abduction or impacted fracture then results (Fig. 6). It is not, of course, suggested that all subcapital fractures are of this stress variety and follow trivial injury. It is presumed that in those caused by single definite injuries there has been a true fracture through healthy bone.

Many patients with subcapital fractures show at first little shortening or lateral rotation of the limb. Within a few hours or days, however, both shortening and rotation are found to increase. The strong retinaculum of Weitbrecht in the posterior capsule is unlikely to be torn in these injuries, but in the anatomical specimen this structure can readily be stripped

FIG. 8

FIG. 9

Figure 8—STAGE III: COMPLETE SUBCAPITAL FRACTURE WITH PARTIAL DISPLACEMENT. The two fragments retain their posterior retinacular attachment, and crushing of the posterior cervical cortex has not yet taken place. Lateral rotation of the distal fragment therefore tilts the capital fragment into abduction and medial rotation as shown radiologically by the direction of the medial weight-bearing lamellae in the femoral head. If the tendency for the limb to rotate laterally is not resisted by external or internal fixation, stripping of the retinacular attachments and crushing of the thin posterior cervical cortex will allow the full displacement of Stage IV to occur.
Figure 9—STAGE IV: COMPLETE SUBCAPITAL FRACTURE WITH FULL DISPLACEMENT. This stage is reached when the retinacular hinge is detached from the posterior surface of the neck and collapse of the posterior cortical shell has taken place. The fragments are then divorced from each other, and the capital fragment at once returns to a more normal position in the acetabulum. Its medial weight-bearing lamellae are then seen radiologically to lie in alignment with their fellows in the pelvis.

from the posterior aspect of the neck (Fig. 10). The tilting of the femoral head in medial rotation and abduction which is so frequently seen in the fresh injury (Fig. 8), suggests that the capital fragment at first retains its posterior attachment to the neck through this retinaculum. The tendency for the limb to rotate laterally will then be resisted by the retinaculum which under the strain will gradually separate from the posterior surface of the neck and allow further deformity to occur. At the same time great pressure will be concentrated on the pivot of the lateral rotation forces at the posterior aspect of the fracture line, with crushing or splintering of the thin posterior cortical shell of the neck. Open reduction of these fractures shows how tightly the fragments are apposed by the surrounding soft tissues, and it is doubtful if full lateral rotation of the distal fragment is possible until at least some

degree of posterior cortical collapse has taken place. When such displacement has occurred the capital fragment is released from the deforming influence of the distal fragment and at once returns to a more normal position in the acetabulum. Its medial trabeculae then lie in alignment with their fellows in the pelvis (Fig. 9).

CLASSIFICATION

Fractures of the femoral neck were at first classified as intracapsular and extracapsular and later distinguished as subcapital, mid-cervical, basal, intertrochanteric or pertrochanteric types. The subcapital type was further subdivided into abduction, or impacted, and adduction, or

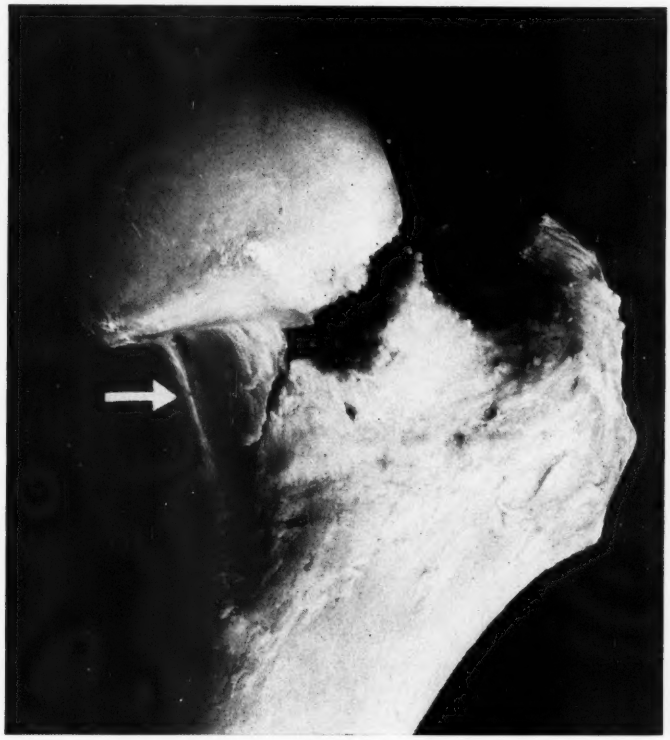


FIG. 10
Subcapital fracture of the femur showing hinge effect of the retinaculum of Weitbrecht in the posterior capsule. After Smith (1953).

varus fractures, but since the classic description by Linton (1949) of the true nature of these fractures this method of subdivision has gradually been discarded in favour of the Pauwels (1935) classification based upon the obliquity of the fracture line as shown in the antero-posterior radiograph. The actual direction of this line, however, remains remarkably constant, and only on rare occasions is a true variation in its obliquity to be found. Subcapital fractures tend to follow the same basic pattern, and their varying radiological appearance is mainly due to the degree of displacement of the fragments. It may sometimes be shown, for example, that an untreated Pauwels Type I fracture will conform to Types II or III as lateral rotation of the distal upon the proximal fragment is allowed to increase with buckling or collapse of the posterior cervical cortex. The Pauwels classification might then appear to be meaningless,

but, in reality, the direction of the fracture line in the antero-posterior radiograph *after* reduction, on which this classification is based, provides a reliable indication of the degree of posterior cortical collapse, and therefore of the limitations of closed reduction. The following classification, which recognises the significance of the radiographic appearances in the varying stages of displacement *before* reduction, is suggested in the hope that it may prove to be of additional value. Stage I: incomplete fracture (Fig. 6). Stage II: complete fracture without displacement (Fig. 7). Stage III: complete fracture with partial displacement (Fig. 8). Stage IV: complete fracture with full displacement (Fig. 9). Intermediary types may sometimes be recognised between the various stages.

REDUCTION

The criteria of reduction in fractures of the femoral neck have never been strictly defined. It is not enough simply to rely upon the impression of poor, fair or satisfactory reduction.

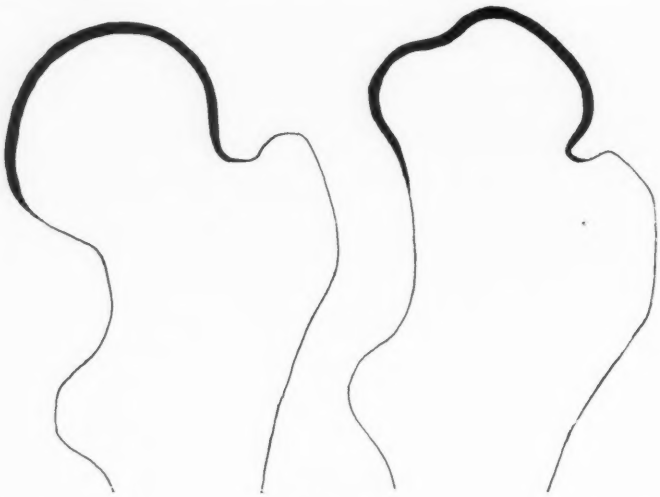


FIG. 11

Orthodiagrams illustrating the shape of the femoral head in the frontal radiograph in undisplaced subcapital fracture (*left*) and subcapital fracture reduced in the extreme valgus position (*right*). Visualisation of the fovea capitis in this view denotes a moderate degree of valgus deformity, but its bitten-out appearance in association with a flattened contour of the articular margin as in the diagram on the right indicates a degree of valgus reposition likely to result in avascular necrosis.

Innumerable examples of "satisfactory" reduction in the literature show, in fact, that adequate reposition has by no means been obtained. The almost spherical shape of the femoral head can be a deceptive factor in the assessment of reduction if its cortical outline alone is considered. On the other hand, the loss of this circular outline is to be regarded as a danger signal (Fig. 11). A true interpretation of the relation of the head to the neck may be obtained by visualising the weight-bearing trabeculae in the two fragments. In the frontal view the medial trabeculae lie at an angle of approximately 160 degrees with the medial femoral cortex. In the lateral view the medial and lateral trabeculae converge and decussate upon an axis which runs in a straight line along the centre of the neck. The normal antero-posterior/lateral alignment may therefore be expressed as 160/180. In this way a rough alignment index of reduction is obtained (Fig. 12). Such factors as distraction, impaction, splintering of the inferior cortex and crushing of the posterior shell, must remain as disturbing contradictions to every claim of perfect reduction, and it becomes increasingly obvious that not only must the fracture be reduced—

it must also be stable. Reduction and stability are interdependent, and herein the solution of the unsolved fracture appears to lie.

In the Stage I subcapital fracture the question of reduction does not arise although the presence of an extreme valgus position, as will shortly be discussed, may cause some misgiving.

In the Stage II fracture gentle handling should dominate its management if troublesome displacement is to be avoided.

In the cleanly broken Stage III fracture reduction is easy and stability is assured. The fragments retain their posterior retinacular attachment, which Smith (1953) has likened to the binding of a book, and it seems important to perform reduction at the earliest opportunity before this binding has been lost. The knee is bent at a right angle and the limb is rotated medially with no attempt to apply other than steadying traction. This manoeuvre should be carried out with extreme gentleness and care, forceful manipulation being reserved for

FIG. 12

Antero-posterior and lateral radiographs to show alignment index of 170/140 in subcapital fracture of the femur. The degree of reduction shown in the lateral view is unacceptable.

the correction of such isolated deformity as persistent forward angulation, which may sometimes be countered by sharply applied medial rotation to the affected limb. While the posterior retinaculum maintains contact between the fragments, traction applied in the long axis of the limb is communicated through it to the posterior aspect of the head. This results in anterior angulation with tilting of the capital fragment. In the same way, any fixation appliance driven across the fracture site while powerful traction is being applied cannot fail to cause tilting of the head. Once reduction of the Stage III fracture has been locked by medial rotation, therefore, compression rather than traction is indicated.

In the Stage IV fracture which has progressed to full displacement the early advantage of continuity between the fragments has been lost, and many of these fractures will defy even the most resolute attempts at both closed and open reduction. Careful pre-operative appraisal of these injuries is advisable in every case, and slipshod radiography should never be accepted. Good radiographs before or during the operation are not only possible but essential, and without them many needless pitfalls will be encountered. Good pre-reduction radiographs will show how frequently this fracture is accompanied by crushing of the thin posterior

cortical shell of the neck and comminution of the inferior cervical cortex, and will indicate how difficult reduction may be. Few surgeons can honestly claim to reconstitute the normal anatomy in these injuries, and it is refreshing to read such forthright observations as that of Cleveland and Fielding (1954) who, in describing the treatment of 335 fractures of the femoral neck, state that "perfect reapposition of the fragments has never been obtained." Stage IV fractures form the hard core of subcapital injuries which are particularly resistant to treatment, and to which the term " unsolved fracture " is undoubtedly meant to apply.

For treatment to be successful this inherently unstable fracture must first be stabilised. This is most easily effected by reduction in the valgus position of lateral rotation deformity to close the crushing defect in the posterior cortex, but the surgeon should recall Pauwels's assertion that the valgus position is nearly always associated with capital necrosis. This assertion must derive considerable support from the recent observations by Smith (1959), which have underlined the importance of preserving the normal relationship of the femoral head in the acetabulum if obliteration of the vessels in the ligamentum teres is to be avoided. This relationship should therefore be maintained in any attempt to secure stability, which may possibly be achieved by the insertion of a wedge-shaped graft to bridge the gap in the posterior cortex, by telescoping the cone-shaped tip of the proximal fragment within the marrow cavity of the neck, by refashioning the fracture surfaces, by corrective osteotomy or by the discovery of new methods of fixation. Further research must obviously be concentrated upon this problem of instability which seems to be the crux of the unsolved fracture. For the time being, the surgeon can do no more than seek stability with every means at his disposal, and try to maintain it by the fixation procedure of his choice. For the latter purpose the use of the low-angle nail has formed the basis of the present inquiry.

OPERATIVE TECHNIQUE

The successful insertion of a low-angle nail depends almost entirely upon the correct location and obliquity of the entry channel in the lateral femoral cortex. If the nail fits this hole snugly, as it should, the direction of the hole will dictate the direction of the nail. In a well reduced and stable fracture with a correctly chosen point of entry the nail is self-locating.

The following technique has been evolved in an attempt to define both the antero-posterior and lateral proclivity of the entry point. Through a lateral incision a 2·5-millimetre marker pin is driven into the outer femoral cortex approximately two and a half inches (6·3 centimetres) below the lower border of the greater trochanter. A metal scale graduated in quarter inches (Fig. 13) is attached to the marker pin and an antero-posterior radiograph is taken. The scale is then removed and a lateral radiograph is taken. The next and most important stage should be undertaken by the surgeon himself despite the delay which a change of gown and gloves entails. A Perspex rule is placed on the antero-posterior film along the line in which the nail is desired to lie—that is, skirting the calcar and aimed towards the centre rather than the summit of the head. The point of entry in relation to the marker pin, together with the length of the nail required, is determined by reference to the shadow of the metal scale on the antero-posterior film. The lateral radiograph is then consulted, and the entry point in this view is again defined by reference to the marker pin.

A quarter-inch hole is now drilled precisely at the point of entry, and a cannulated drill-reamer is used in a hand-chuck to enlarge the hole and convert it into an oblique channel. The correct degree of obliquity is found by advancing the reamer from time to time until it is found to slide firmly upwards along the buttress of the calcar. With the reamer still in place, a guide wire may be inserted to check the accuracy of the entry channel in both views. If the entry channel is correct, the reamer is removed and a nail of the appropriate length inserted. Some resistance will at first be felt as the nail firmly negotiates the calcar femorale and inferior buttress of the neck.

TABLE I

ANALYSIS OF THIRTY-ONE PATIENTS WITH TROCHANTERIC, BASAL AND MID-CERVICAL FRACTURES. ALL FRACTURES UNITED

Case number	Age (*years*)	Sex	Type of fracture	Period before operation (*days*)	Alignment index	Period before weight bearing (*days*)	Period in hospital (*days*)	Period of follow-up (*months*)
1	86	Female	Pertrochanteric	12	160/180	2	40	16
2	78	Male	Pertrochanteric	14	170/180	1	54	6
3	66	Male	Pertrochanteric	4	160/180	1	30	65
4	60	Female	Pertrochanteric	4	165/180	1	35	64
5	67	Female	Pertrochanteric	2	150/180	2	33	64
6	79	Female	Pertrochanteric	5	160/180	10	60	63
7	59	Female	Intertrochanteric	3	170/180	2	27	62
8	70	Female	Basal	4	160/180	30	20	54
9	79	Female	Intertrochanteric	1	160/180	2	11	52
10	69	Female	Pertrochanteric	7	160/180	10	57	19
11	71	Female	Pertrochanteric	9	155/180	5	41	51
12	74	Female	Basal	2	160/180	3	13	10
13	71	Female	Intertrochanteric	4	160/180	7	50	51
14	62	Female	Mid-cervical	7	170/170	1	17	50
15	74	Male	Pertrochanteric	5	160/180	42	94	49
16	58	Male	Basal	3	165/180	5	14	18
17	80	Female	Pertrochanteric	4	170/180	10	47	10
18	71	Female	Intertrochanteric	3	170/170	30	59	46
19	78	Female	Pertrochanteric	1	160/180	1	30	45
20	69	Female	Intertrochanteric	2	160/170	2	20	44
21	80	Female	Pertrochanteric	1	165/160	4	10	44
22	62	Female	Intertrochanteric	3	160/150	—	69	42
23	70	Female	Pertrochanteric	3	155/170	4	32	42
24	68	Male	Intertrochanteric	3	160/170	3	38	14
25	84	Female	Intertrochanteric	1	160/165	1	14	39
26	78	Female	Basal	3	170/165	4	19	33
27	53	Female	Basal	4	160/180	15	35	33
28	73	Male	Pertrochanteric	4	160/180	4	74	32
29	82	Female	Mid-cervical	5	160/180	20	135	21
30	61	Female	Intertrochanteric	4	160/180	10	60	21
31	54	Male	Intertrochanteric	10	160/180	20	29	21

TABLE II

ANALYSIS OF EIGHTY PATIENTS WITH SUBCAPITAL FRACTURES SURVIVING FOR TWELVE MONTHS OR LONGER

Case number	Age (*years*)	Sex	Stage of fracture	Period before operation (*days*)	Alignment index	Period before weight bearing (*days*)	Period in hospital (*days*)	Period of follow-up (*months*)	Result
32	60	Female	IV	4	185/180	4	34	65	Union*
33	64	Female	IV	4	160/180	12	44	29	Union
34	69	Female	IV	5	160/165	6	37	64	Non-union
35	72	Female	III	2	190/180	1	17	64	Union*
36	74	Female	IV	1	160/165	6	24	63	Non-union
37	74	Female	II	2	170/175	5	25	63	Union
38	82	Female	I	1	170/165	3	13	13	Union
39	86	Female	I	4	170/170	3	22	12	Union
40	67	Female	III	4	160/170	1	22	61	Union
41	66	Female	III	2	200/180	3	44	60	Union*
42	75	Female	IV	6	175/150	7	22	57	Non-union
43	52	Female	III	5	190/175	1	16	56	Union*
44	60	Female	IV	4	150/140	2	19	56	Non-union
45	66	Female	IV	1	160/150	—	62	56	Non-union
46	75	Female	IV	5	160/180	2	17	55	Union
47	57	Female	III	1	200/150	6	27	55	Union*
48	62	Female	IV	1	160/180	1	24	51	Union
49	72	Female	I	10	170/180	1	21	51	Union
50	81	Female	III	1	185/170	2	27	50	Union*
51	47	Female	IV	3	100/140	7	13	50	Non-union
52	90	Female	I	1	180/170	3	20	14	Union
53	73	Female	IV	2	170/160	2	30	14	Union
54	65	Female	III	1	180/170	1	17	49	Union
55	74	Female	I	4	180/180	1	57	46	Union
56	70	Female	IV	3	155/150	7	49	43	Non-union
57	86	Female	IV	4	165/180	1	25	48	Union
58	67	Female	III	2	165/180	3	26	23	Union
59	69	Female	III	5	160/180	—	35	47	Non-union
60	75	Female	III	1	170/170	3	38	45	Union
61	67	Female	III	2	160/170	10	20	45	Union*
62	67	Male	IV	2	180/170	5	40	45	Union
63	64	Female	IV	1	200/180	1	15	45	Union*

* Avascular necrosis.

TABLE II—*continued*

ANALYSIS OF EIGHTY PATIENTS WITH SUBCAPITAL FRACTURES SURVIVING FOR TWELVE MONTHS OR LONGER

Case number	Age (*years*)	Sex	Stage of fracture	Period before operation (*days*)	Alignment index	Period before weight bearing (*days*)	Period in hospital (*days*)	Period of follow-up (*months*)	Result
64	46	Male	IV	1	180/170	1	21	45	Union
65	50	Female	II	9	180/170	2	20	44	Union
66	69	Female	IV	2	180/130	21	30	15	Non-union
67	56	Female	IV	3	170/140	5	32	42	Non-union
68	67	Female	IV	2	180/140	14	18	42	Non-union
69	69	Female	III	4	160/180	5	40	15	Union
70	69	Female	III	7	190/160	1	60	39	Union*
71	65	Female	II	10	170/180	2	28	38	Union
72	74	Female	IV	2	170/170	1	50	38	Union
73	62	Male	III	11	180/165	3	28	38	Union
74	58	Female	II	6	165/160	9	55	37	Union
75	70	Female	III	5	150/160	—	30	37	Non-union
76	78	Female	III	6	160/180	7	25	34	Union
77	66	Female	III	2	160/180	3	22	33	Union
78	63	Female	III	2	200/180	2	22	31	Union*
79	40	Male	II	2	160/180	2	17	30	Union
80	51	Male	IV	5	160/160	3	15	30	Union
81	56	Male	II	2	180/180	1	12	28	Union
82	76	Female	III	5	160/180	8	11	28	Union
83	52	Female	IV	3	180/150	15	21	27	Non-union
84	63	Female	II	3	160/180	3	48	27	Union†
85	67	Female	I	7	160/180	2	15	26	Union†
86	70	Female	IV	2	160/160	6	9	26	Union
87	83	Female	III	2	180/180	6	16	25	Union
88	74	Female	IV	9	170/180	8	58	24	Non-union
89	66	Female	I	3	160/180	6	20	23	Union†
90	64	Female	III	2	170/180	5	14	23	Union
91	66	Male	II	3	160/180	5	26	22	Union
92	71	Male	II	2	160/180	18	54	21	Union
93	59	Female	III	3	160/155	4	38	19	Union†
94	75	Female	IV	1	160/160	7	30	19	Union†
95	72	Female	III	2	160/170	6	14	18	Union

* Avascular necrosis. † Low-angle screw.

TABLE II—*continued*

ANALYSIS OF EIGHTY PATIENTS WITH SUBCAPITAL FRACTURES SURVIVING FOR TWELVE MONTHS OR LONGER

Case number	Age *(years)*	Sex	Stage of fracture	Period before operation *(days)*	Alignment index	Period before weight bearing *(days)*	Period in hospital *(days)*	Period of follow-up *(months)*	Result
96	60	Male	III	4	160/180	5	20	18	Union†
97	86	Female	IV	3	160/180	7	21	17	Union
98	72	Female	III	6	160/180	5	30	16	Union
99	74	Female	III	2	160/180	8	27	16	Union†
100	34	Male	III	2	160/180	5	28	16	Union†
101	51	Male	II	1	160/180	2	15	16	Union†
102	51	Female	IV	4	165/180	2	18	15	Union
103	70	Female	I	5	170/180	3	17	15	Union
104	38	Male	III	2	165/160	10	17	15	Union
105	70	Female	III	4	160/180	4	46	15	Union
106	64	Female	III	4	195/170	14	35	14	Union*
107	62	Female	IV	1	175/155	3	18	14	Non-union
108	56	Female	I	1	160/180	1	25	13	Union
109	39	Male	II	5	160/180	2	16	13	Union†
110	72	Female	IV	2	170/160	6	49	13	Union†
111	73	Female	IV	6	170/180	17	39	12	Union†

* Avascular necrosis. † Low-angle screw.

This brief description of the operative technique is by no means intended to convey the impression that low-angle nailing is always a straightforward procedure. On the contrary, it is often a most difficult undertaking, and only by meticulous attention to detail can it be expected to succeed.

MATERIAL

The low-angle Küntscher nail is unsuitable for the treatment of fractures involving the lesser trochanter with fracture of the calcar femorale at this level. Without the support of the calcar the nail inclines inwards until it lies against the medial femoral cortex below the line of fracture and varus deformity occurs. During the past five and a half years 450 patients with all types of fractures of the femoral neck have been admitted to the Preston Royal Infirmary. One hundred and fifty-nine of these fractures were situated above the level of the lesser trochanter, and were considered suitable for low-angle fixation. Full weight bearing was encouraged whenever possible after operation, and many patients were walking unaided and discharged home within a few weeks of their injury.

RESULTS

Thirty-one patients who sustained mid-cervical, basal, intertrochanteric or high pertrochanteric fractures, and who survived for at least six months, all obtained full union (Table I). These fractures unite freely with any modern method of treatment, but consolidation

is undoubtedly hastened by early weight bearing. In these particular injuries, therefore, the advantages of low-angle fixation are self-evident.

The assessment of union in subcapital fractures within twelve months of the injury is generally considered unwise. Of eighty patients in this series who were under observation for twelve months or longer sixty-five (81 per cent) showed full radiological union (Table II).

TABLE III

SUBCAPITAL FRACTURES: RELATION OF UNION TO STAGE OF FRACTURE

Stage	Number of fractures	United	Ununited	Percentage uniting
I	9	9	—	100
II	11	11	—	100
III	30	28	2	93
IV	30	17	13	57
Totals	80	65	15	81

On closer analysis the problems of union were seen to arise almost exclusively in the Stage IV fractures, of which only 57 per cent united (Table III). It is therefore misleading to present the results of treatment in subcapital fractures as an overall percentage, and the value of the low-angle, or any other, fixation procedure should be judged principally by the results of its use in fully displaced Stage IV injuries.

Examination of the relationship between reduction and union shows that non-union in this series was largely confined to those cases in which an alignment index of less than 155 was accepted in either the antero-posterior or lateral view. It is clear that poor reduction is almost synonymous with non-union, and good reduction greatly improves the chances of success. Low-angle fixation after imperfect reduction may then be regarded as a worthless procedure.

COMPLICATIONS

Movement of the nail—The Küntscher clover-leaf type of nail has the advantage of strength and lightness, but cannot always be relied upon to remain in place. In several instances the nail intruded into the hip joint or extruded sufficiently to allow slipping at the fracture site. For this reason a low-angle screw with a short self-tapping thread has been devised (Fig. 13, D). The capital fragment alone is held by the screw, and its shank can extrude as need be through the sleeve formed by the entry channel in the lateral femoral cortex. In the stable fracture impaction is ensured in this way as absorption or settling at the fracture site occurs. It is now believed, however, that in the fully displaced unstable fracture such " absorption or settling " is often mistaken for an increasing collapse of the posterior cortex with the return of anterior angulation and breakdown of reduction. As a consequence, the fallacy of overall compression in the treatment of displaced subcapital fractures has been recognised. The screw, now under trial, has therefore been provided with a longer thread to engage both fragments, and, when positioned posteriorly, to maintain distraction at the posterior aspect of the fracture (Fig. 13, E). Since the capital fragment is mobile, distraction posteriorly results in compression anteriorly. In this sense, distraction really implies restoration of the normal compressive function of the posterior cervical cortex.

Avascular necrosis—No discussion of femoral neck fractures can escape the question of avascular necrosis, especially one in which early weight bearing is being advised. According to d'Aubigné (1958) 50 per cent of all subcapital fractures develop avascular necrosis. In this

series complete collapse and disappearance of the head was seen once, and well defined avascular change with localised collapse was seen in ten fractures which united.

Even with severe disruption of the retinacular vessels, the foveal artery, as Tucker (1949) has shown, remains as a source of nutrition in most elderly patients. In this connection it is of the utmost significance to record that avascular necrosis followed union in every case in which an alignment index greater than 180 in the frontal view—in other words an extreme valgus position—was accepted at reduction in this series. It is equally significant that this complication was encountered only once when the alignment index was less than 180, and this in a case where the nail entered the hip joint. Both in regard to union and to avascular necrosis, therefore, the safe limit of malposition appears to lie within the narrow alignment index range of 155/180 in the antero-posterior and 180/155 in the lateral radiograph.

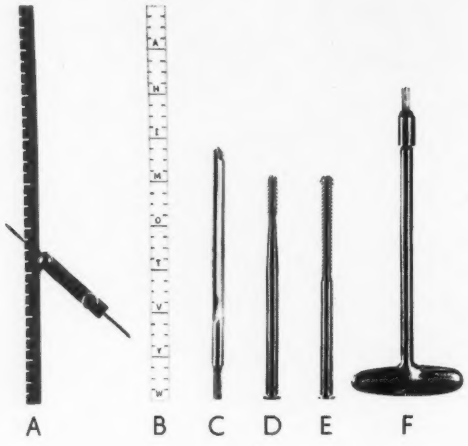


FIG. 13

A—Metal measuring scale with marker pin. B—Perspex rule. C—Cannulated drill-reamer. D—Cannulated low-angle screw with short self-tapping thread. E—Cannulated low-angle screw with extended self-tapping thread. F—Cannulated Allan key driver.

Degenerative arthritis—In regard to the late results in the series under examination it is expected that even the restricted limits of malposition defined above may prove to be over generous. Failure to achieve perfect reduction will at the same time fail to restore the critical relationship of the femoral head in the acetabulum with which it is congruous only in the position of weight bearing (Walmsley 1928). The constant apposition of dissimilar articular surfaces, which will then ensue, must inevitably lead to degenerative arthritic changes.

SUMMARY AND CONCLUSIONS

The successful management of femoral neck fractures is obviously based upon many factors. The forces acting upon the proximal end of the femur are believed to be mainly compressive in nature, and the low-angle nail by stabilising the *fully reduced* fracture in the line of these forces is held to allow weight bearing to take place. Low-angle nailing is believed to offer many advantages over conventional methods of treatment but only in the presence of stability. Stable reduction is the essential preliminary to any form of treatment, and low-angle fixation with early weight bearing in the absence of stability is regarded as futile.

It is suggested that those subcapital separations which follow trivial injury may originate as stress fractures accompanying the process of bone remodelling in the aged, and that many of these fractures may remain unrecognised and heal spontaneously. With rare exceptions, subcapital fractures are regarded as being of the same essential pattern, and their varying radiological appearance is considered to be due to the different degrees of displacement to which they have been subjected. A new classification based on this premise has been suggested.

In a series of eighty subcapital fractures the incidence of avascular necrosis was not adversely affected by early weight bearing, but reduction in the extreme valgus position was invariably followed by this disaster. This is probably also true of any malposition in extreme rotation which must stretch and obliterate the vessels in the ligamentum teres.

A rough alignment index of reduction was found to provide an almost infallible guide to the prognosis both in regard to union and to avascular change. It may therefore be possible to base prognosis on the quality of reduction *before the fixation appliance has been inserted.* The unsatisfactory results in those cases apparently destined to non-union or avascular necrosis may then be avoided by alternative means of treatment at an early stage. Whether this will prove to be true must depend upon a much longer experience of low-angle fixation, and, in common with almost every communication on this subject, premature publication must largely offset the value of the present findings.

I am much indebted to Mr N. A. G. Covell and to my registrars Mr F. F. Silk and Mr D. G. Wray for their encouragement, assistance and invaluable criticism in the work on which this inquiry has been based.

REFERENCES

D'AUBIGNÉ, R. Merle (1958): Pseudarthroses du col du fémur. *Presse Médicale*, **66,** 813.

BACKMAN, Stig (1957): The Proximal End of the Femur. *Acta Radiologica*, Supplementum 146.

BRITTAIN, H. A. (1942): The Low Nail. *British Medical Journal*, **i,** 463.

BURNS, B. H., and YOUNG, R. H. (1944): Early Movement in the Treatment of Closed Fractures. *Lancet*, **i,** 723.

CLEVELAND, M., and FIELDING, W. J. (1954): A Continuing End-result Study of Intracapsular Fracture of the Neck of the Femur. *Journal of Bone and Joint Surgery*, **36–A,** 1,023.

DICKSON, J. A. (1953): The " Unsolved " Fracture. *Journal of Bone and Joint Surgery*, **35–A,** 805.

FARKAS, A., WILSON, M. J., and HAYNER, J. C. (1948): An Anatomical Study of the Mechanics, Pathology, and Healing of Fracture of the Femoral Neck. *Journal of Bone and Joint Surgery*, **30–A,** 53.

GARDEN, R. S. (1961): The Structure and Function of the Proximal End of the Femur. *Journal of Bone and Joint Surgery*, **43–B,** 576.

GODOY MOREIRA, F. E., and CAMARGO, F. P. de (1957): Contribuição ao tratamento das fraturas do colo do femur. *Revista do Hospital das Clínicas*, **12,** 73.

KING, T. (1939): The Closed Operation for Intracapsular Fracture of the Neck of the Femur. *British Journal of Surgery*, **26,** 721.

KÜNTSCHER, G. (1953): Die vollautomatische Schenkelhalsnagelung. *Zeitschrift für Orthopädie und ihre Grenzgebiete*, **84,** 17.

LINTON, Per. (1949): Types of Displacement in Fractures of the Femoral Neck. *Journal of Bone and Joint Surgery*, **31–B,** 184.

PATRICK, J. (1949): Intracapsular Fractures of the Femur Treated with a Combined Smith-Petersen Nail and Fibular Graft. *Journal of Bone and Joint Surgery*, **31–A,** 67.

PAUWELS, F. (1935): *Der Schenkelhalsbruch. Ein mechanisches Problem.* Stuttgart: Ferdinand Enke.

SMITH, F. B. (1959): Effects of Rotatory and Valgus Malpositions on Blood Supply to the Femoral Head. *Journal of Bone and Joint Surgery*, **41–A,** 800.

SMITH, L. D. (1953): Hip Fractures. *Journal of Bone and Joint Surgery*, **35–A,** 367.

TUCKER, F. R. (1949): Arterial Supply to the Femoral Head and its Clinical Importance. *Journal of Bone and Joint Surgery*, **31–B,** 82.

WALMSLEY, T. (1928): The Articular Mechanism of the Diarthroses. *Journal of Bone and Joint Surgery*, **10,** 40.

DELAYED OPERATION IN THE OPEN REDUCTION OF FRACTURES OF LONG BONES

JOHN CHARNLEY, MANCHESTER, ENGLAND, and ADLY GUINDY, CAIRO, EGYPT

From Park Hospital, Davyhulme, Manchester

Murray (1941) reviewed evidence for the opinion that the best time to carry out internal fixation of a fracture is as soon after the injury as possible. The advantages claimed for immediate operation are the minimal interference with the normal healing process of a fracture, the ease of exposure and reduction, and the accurate identification of damaged tissues unaltered by oedema and fibrosis.

In 1959 Smith recorded 130 fractures of the radius and ulna in adults treated by internal fixation. Fifty-two fractures treated by delayed internal fixation showed less delay in union than did the remaining seventy-eight fractures operated upon early. There was in fact no case of true non-union in the patients subjected to operation after a delay of one week, whereas there was an incidence of 21·8 per cent of non-union when the fractures were operated upon early. He concluded that the incidence of non-union after internal fixation can be reduced if operative treatment is delayed for one week, and preferably for two or three weeks, after the injury.

Smith and Sage (1957) reported results which were at variance with the findings of Smith. In a series of 253 fractures treated by medullary nailing Smith and Sage found 19·7 per cent of non-union in fractures nailed within fourteen days of injury, 19·3 per cent of non-union in those nailed fifteen to ninety days after injury, and 30·1 per cent of non-union in those nailed three months or more after the injury.

In view of this discrepancy it was decided to investigate again the relationship between delayed operation and non-union as illustrated by the results of intramedullary fixation of fractures of the shaft of the femur.

MATERIAL

The material consisted of thirty-seven patients with thirty-eight fractures of the shaft of the femur. Thirty-six fractures were closed and two were compound but healed without bone infection.

In all patients a Küntscher nail of clover-leaf pattern was inserted by the retrograde method after open exposure of the fracture. Almost all these cases were operated upon by the same surgeon (J. C.). The review covers twelve years from 1948 to 1960.

METHOD OF INVESTIGATION

Assessment of the time required to obtain union—To assess osseous union after intramedullary fixation one is entirely dependent on radiological tests because the rigidity conferred by the nail makes clinical testing impossible. All patients in this study were radiographed approximately every month in the early period and every three months in the later period, and radiographic examinations were continued until union was definitely seen.

The criterion for osseous union was the presence of continuous bridging of bone at some part of the fracture, though not necessarily on all sides, combined with the absence of sclerosis. If sclerosis was present and the texture of the bone at the fracture site was not clearly revealed, a decision on the state of the union was postponed until further radiographic examination had been made three months later, and repeated again if necessary in the same way, until the sclerosis was diminishing and a bridge of bone was quite clearly visible.

In general, osseous union was proved by progressive improvement of the radiological picture after weight bearing had been started. When the radiological picture was not improving six months or more after the operation, in three cases, the fracture was explored for the purpose of applying a prophylactic bone graft, and the fracture was thereby proved to be ununited. In another three patients a spontaneous fracture of the nail drew attention to the non-union, and in one the femur immediately fractured again after removal of the nail which was made necessary by pain caused by excessive projection of the proximal end through the trochanter (Table I).

TABLE I

ANALYSIS OF PATIENTS WITH NON-UNION

Case number	Age (*years*)	Direction and site of fracture	Interval (*days*)	Radiographic appearance after three months	Callus value	Radiographic appearance after six months	Further state until grafting was done
1	36	Transverse mid-shaft	1	No callus bridging the fracture	0	No callus bridging	Still no callus bridging at nine months; grafted after eleven months. Nail was replaced—fracture was immediately loose
2	42	Slightly oblique mid-shaft	1	Slight callus on medial side	20	No change	No evidence of union after ten months. Grafted after eleven months. Fracture found ununited at operation
3	30	Transverse mid-shaft	3	—	—	—	Non-union after eight and a half months. Fatigue fracture of nail at ten months. Pseudarthrosis
4	46	Transverse mid-shaft	5	Faint callus on one side (medial)	32	No change	Fatigue fracture of nail after fifteen months. Ends of fracture freely mobile
5	33	Oblique mid-shaft	1	Only small bridge medially. Ischaemia about fracture on lateral aspect	20	No change—still ischaemic and fluffy	Bone grafting done after six months. Only small bridge of bone medially
6	72	Slight oblique mid-shaft	1	Small amount of callus anteriorly and doubtful if really bridging	120	No change	Nail removed because of pain in hip at seven months. Immediate refracture. Grafted and nail reinserted
7	28	Transverse mid-shaft	100	Slight callus	15	No change	Eighteen months: non-union with sclerosis of ends. Partial fracture of nail. Twenty-two months: nail partially fractured. Grafted and new nail inserted

It is possible to argue that two of these cases were not truly cases of non-union and should more accurately be described as cases of refracture. From a practical point of view, however, they were both cases of defective consolidation in which a tenuous bridge of bone refused to hypertrophy in a time considerably longer than is necessary for consolidation in the normal process of fracture union.

Amount of callus—As a side issue on the relationship between the time elapsing between injury and internal fixation we tried to assess whether the volume of periosteal callus was noticeably different after different intervals of delay.

The amount of callus was estimated by making tracings direct from a radiograph and measuring the surface area on a transparent grid having 144 small squares to the square inch.

The grid was superimposed on the tracing of a radiograph, and the area occupied by the callus was counted on the grid and called the " callus value." No account was taken of the internal architecture of the callus, or of variations of its density, and the total area enclosed by the external outline of the callus was the criterion of the amount of callus. The radiological view which showed the greater amount of callus, usually the antero-posterior, was the one used for assessment.

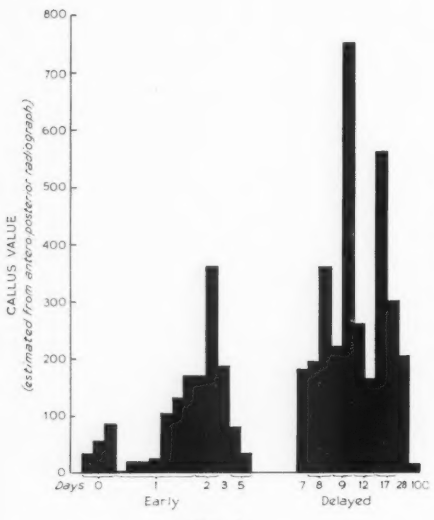


FIG. 1

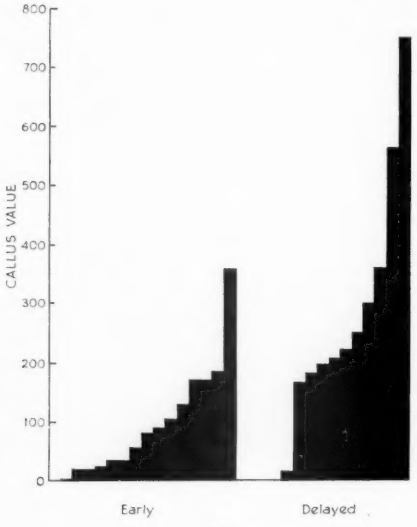


FIG. 2

Figure 1—Histogram indicating the amount of callus in cases arranged in order of duration of the interval between injury and operation. Figure 2—Histogram using the same material as in Figure 1 but with cases arranged in ascending order of callus value in the two groups " early " and " delayed."

The callus value was not estimated before the end of the first three months. It was hoped in this way to express the volume of consolidating callus rather than what might be the greater volume of the cloud or haze of early callus which is often present six weeks after a fracture of the femoral shaft in young adult patients.

RESULTS

Osseous union—Of the thirty-eight fractures, twenty-four were treated by nailing between one day and six days after injury, and fourteen by nailing between seven and twenty-eight days after injury. In one patient the fracture was nailed 100 days after injury without any bone graft being used.

The cause of delay between injury and operation was either that conservative treatment had been tried and had failed to achieve an acceptable position, or because the general condition of the patient with multiple injuries was unsuitable for immediate surgery. In these latter, once the emergency treatment had been completed, internal fixation of the femur was postponed till a convenient operation session.

Of the twenty-four fractures treated by nailing between one and six days after injury six failed to unite and were caused to unite only after bone grafting—an incidence of non-union of 25 per cent. The rest (eighteen patients) were discharged with osseous union proved radiologically after periods varying from four and a half to eighteen months.

Of the fourteen fractures treated by nailing seven or more days after injury only one failed to unite—namely, the one that was nailed after 100 days. The remaining thirteen all showed radiographic evidence of solid union in a period varying from four to thirteen months and the patients were discharged within that period (Table II).

Amount of callus—The amount of callus three months after operation showed a striking difference in the patients operated on within six days of the injury and those operated on

TABLE II

COMPARISON OF RESULTS WITH EARLY AND DELAYED TREATMENT

Type of treatment	Number of fractures	Number with non-union	Percentage
Early operation (0–6 days) .	24	6	25
Delayed operation (7 days +)	14	1	7·14

TABLE III

AGE FACTOR IN PATIENTS TREATED WITH EARLY AND DELAYED OPERATION

Age (*years*)	Early operation (*patients*)	Delayed operation (*patients*)
15–19	4	1
20–29	5	7
30–39	3	1
40–49	4	2
50–59	2	—
60–69	3	2
70–75	3	—
Total patients	24	13
Total fractures	24	14

TABLE IV

SITE OF FRACTURES TREATED

Site of fracture	Early operation (*fractures*)	Delayed operation (*fractures*)
Junction of uppermost and middle thirds	5	1
Mid-shaft	18	11
Junction of lowest and middle thirds .	1	2

ıfter that period. In the early group the range of callus values was from 0–185 units, with ›nly one exception. The exception had a callus value of 360 units in a spiral fracture involving ılmost half the length of the shaft. In the delayed group the callus value ranged from 185 o 750, with one exception. The exception had a callus value of 15 units and was the case ›perated upon and nailed 100 days after injury, and this resulted in non-union.

When the cases within each group were compared with each other it was seen that the callu value did not necessarily increase in direct proportion to the number of days elapsing betwee the fracture and the operative fixation. This feature is brought out in alternative arrangement of the same data in the histograms of Figures 1 and 2.

A brief check was made of other factors—such as the degree of displacement, the age o the patient and the site of fracture—which might have a bearing on union (Tables III and IV) All these fractures which had been selected for intramedullary nailing showed a full diamete

FIG. 3

FIG. 4

Figure 3—Radiograph of right femur showing fracture united but with varus deformity. Figure 4—Radiograph of left femur showing sound union.

displacement in the pre-operative radiographs, because in this clinic cases with less displacement are routinely treated by conservative means; so it may be presumed that all the fractures in this series were associated with considerable soft-tissue damage.

Six of the seven patients with fractures ending in non-union were aged from twenty-eight to forty-six years; only one was seventy-two years old. Five were men and two women.

All the fractures that showed non-union were situated in the middle third of the shaft. Four fractures were transverse and three slightly oblique.

One case is considered worthy of description in considerable detail because it provides the opportunity to compare the healing of two femoral shaft fractures sustained at the same time in the same patient and in whom one fracture was nailed on the day of the accident and the other twelve days later. Unfortunately, the mechanical efficacy of the internal fixation was not exactly the same in both femora, and delay in consolidation on one side could be due

result of less perfect fixation on that side. The fact that the side of defective consolidation was operated upon on the same day as the injury might therefore be pure coincidence, but nevertheless the case merits description.

CASE REPORT

A man aged twenty-two years sustained closed fractures of both femora at nearly the same level (junction of middle and lowest thirds) in a traffic accident in October 1951, at the same time receiving a compound fracture of the left tibia and a fracture of the neck of the right mandible. The right femur was nailed on the day of the accident. The fixation was not absolutely rigid; so the limb was supported by external fixation in a Thomas's splint for twenty-five days. At the same time the left tibia was plated. The fracture of the left femur was treated by traction on a Thomas's splint.

Under traction the position of the fracture of the left femur remained unsatisfactory, and it was nailed twelve days after the accident and twelve days later than the right femur. This time the rigidity of fixation was very good and no external support was considered necessary.

Progress. *Right femur*—After three months the callus value was 120 and a varus deformity of 5 degrees developed. After four and a half months the callus value was 294 and the varus deformity had increased and become detectable clinically. The patient complained of pain in the lower end of the thigh on walking. When seen two years after the accident the fracture was solidly united (Fig. 3). *Left femur*—After three months the alignment was good and union was progressing; at four months the callus value was 240 and sound bridging by callus was evident. Weight bearing was permitted and union thereafter was uneventful (Fig. 4).

Comment—Although neither fracture failed to unite in the end, and quite good callus values were present on both sides, the fact is that the fractured femur treated by early operation was slow in consolidating whereas the fracture on the opposite side treated by operation after twelve days united excellently.

It is true that a mechanical factor may have contributed to the difference in rate of union of the fractures of the right and left femora; on the right side the intramedullary nail reached only three and a half inches below the fracture line compared with nearly five inches on the left side; but the right limb was fixed post-operatively for nearly four weeks on a Thomas's splint to reinforce the fixation. Though not conclusive, it would seem not unreasonable to suggest that early operative intervention in a youth aged twenty-two years depressed the power of union on the right side, but that the delay of operative intervention on the left side enabled union to proceed in the same vigorous way that one would expect under conservative treatment in a young patient.

DISCUSSION

Comparison of the results of early and delayed internal fixation in this series shows that the incidence of non-union in the early group was 25 per cent and 7 per cent in the delayed group. The isolated instance of non-union in the delayed group will receive special comment because the delay was as long as 100 days, and it can be argued that this might justify its exclusion from the series, in which case the difference in the rate of non-union between the early and late cases would be as 25 per cent to nil.

An explanation of the difference in the rate of osseous union after early and late internal fixation might lie in a difference in the circulation of the blood in the ends of a fractured bone when the operation is done early or late. When a fracture is exposed under a tourniquet within a few days of injury the ends of the bones have the appearance of ivory cortical bone which is almost as white as that seen in a cadaver. When a fracture is exposed some weeks after injury, also under a tourniquet, the bones vary in colour from pink to a deep maroon as a result of the enlargement of Haversian spaces in the cortical bone. In some cases when operation is undertaken for non-union there is a detectable difference in colour between the two fragments, one being pale and the other maroon. In other cases a sharp demarcation may exist between a pale part of the bone, usually at the fractured end of one fragment, and the maroon-coloured bone with which it is in continuity.

It is becoming increasingly recognised that when a long bone is fractured the longitudina circulation of the Haversian systems cannot continue at the broken surface of cortical bon which presents to the fracture-haematoma. When a fracture is left to unite under conservativ treatment the ends of the bones eventually take part in the general hyperaemia of the fracture though enlargement of the Haversian systems in the ends of the fragments will lag behin the enlargement of the Haversian systems in undamaged parts of the bone. If th periosteum is stripped near the ends of the bones in a fresh fracture it seems probable tha the start of hyperaemia in the ends of the bone is delayed artificially and that in the condition of a fresh fracture the reposition of soft parts against the denuded ends of the bone does no secure entry of blood vessels in sufficient number to cause hyperaemia. When operativ intervention is delayed until the ends of the bones have started to take part in the hyperaemi process, it seems possible that the bone ends do not suffer similar ischaemia or, if they ar rendered ischaemic at the moment of operation, that this is only temporary and the precedin hyperaemia renders them capable of recovering a good supply very quickly.

In this connection figures for the " callus value " of femoral fractures studied in thi paper three months after operation are of interest because it cannot be denied that profus callus formation must be linked with a good blood supply. The average amount of callu measured three months after the operation, on the whole was greater in the cases in thi series which had been operated on after a delay of six or more days than in those operated o after one to six days.

The potential for osseous repair which can be evoked from bone by a second mechanical insult some weeks or months after a fracture that is showing delayed union is evident in those methods of treating non-union which do not rely primarily on a bone graft. Naughton Dunn (1939) treated non-union of the tibia merely by " shingling " the bone ends without disturbing the fibrous intersection at the site of the fracture. Phemister (1947) did more or less the same with the addition of an autogenous free graft laid on the side of the fracture, and Jarry and Uhthoff (1960) in their " petal " technique have recently drawn attention to the latent powers of osteogenesis which can be liberated in an ununited fracture by mechanical trauma.

It seems reasonable to suggest, therefore, that delayed internal fixation may be beneficial in two quite different ways. Firstly, it is beneficial in that it withholds the harmful effects of early intervention, which tends to increase the volume of ischaemic cortical bone in any fresh fracture (Urist, Mazet and McLean 1954). Secondly, delayed internal fixation may be beneficial in applying a second stimulus for osteogenesis in fractures which are in imminent danger of passing into delayed union. When delayed union is threatened one can visualise the ends of the bones becoming invested in fibrous tissue, with a danger that they may become sealed off and insulated from each other. The addition of more mechanical trauma at this stage may break down the process of investment at a critical phase and thus precipitate union.

This line of thought would be compatible with the occasional experience after open reduction and internal fixation which has been delayed for four or five weeks, and when no bone graft is used, that the line of the fracture will become radiographically invisible with surprising speed and remarkable completeness, and in these cases the bone is perceptibly porotic in comparison with a fresh fracture.

The significance of the solitary case of non-union which occurred in a series of patients subjected to delayed primary intervention demands special comment. In this solitary failure the interval between injury and internal fixation was 100 days. (It is not stated in the case records why a bone graft was not used as an adjunct to internal fixation in this instance, as would be the normal procedure in this clinic.) The failure to obtain osseous union might indicate that after 100 days the original stimulus to union caused by the fracture had been lost, that traumatic hyperaemia after this time had disappeared, and that the mechanical insult of internal fixation at such a late date should be regarded not as delayed intervention but as a primary intervention on quiescent bone.

SUMMARY AND CONCLUSIONS

1. The findings in this series of fractures of the shaft of the femur treated by intramedullary nailing confirm the observation of Smith (1959) that the incidence of non-union is significantly diminished when operative intervention is postponed more than one week from the time of the injury.

2. The series is too small to afford conclusive proof, and it offers no explanation of the opposite findings of Smith and Sage (1957), but it indicates that this very important aspect of the operative treatment of fractures ought to be submitted to thorough investigation by many more observers.

REFERENCES

DUNN, N. (1939): Treatment of Ununited Fractures. *British Medical Journal*, **ii,** 221.

JARRY, L., and UHTHOFF, H. K. (1960): Activation of Osteogenesis by the " Petal " Technique. *Journal of Bone and Joint Surgery*, **42–B,** 126.

MURRAY, C. R. (1941): The Timing of the Fracture-healing Process. *Journal of Bone and Joint Surgery*, **23,** 598.

PHEMISTER, D. B. (1947): Treatment of Ununited Fractures by Onlay Bone Grafts Without Screw or Tie Fixation and Without Breaking Down of the Fibrous Union. *Journal of Bone and Joint Surgery*, **29,** 946.

SMITH, H., and SAGE, F. P. (1957): Medullary Fixation of Forearm Fractures. *Journal of Bone and Joint Surgery*, **39–A,** 91.

SMITH, J. E. M. (1959): Internal Fixation in the Treatment of Fractures of the Shafts of the Radius and Ulna in Adults. *Journal of Bone and Joint Surgery*, **41–B,** 122.

URIST, M. R., MAZET, R., Jun., and MCLEAN, F. C. (1954): The Pathogenesis and Treatment of Delayed Union and Non-union. *Journal of Bone and Joint Surgery*, **36–A,** 931.

SUBCORTICAL ILIAC BONE GRAFTS IN FRACTURE OF THE TIBIA

D. B. FORBES, MONTREAL, QUEBEC, CANADA

From Park Hospital, Davyhulme, Manchester, England

This is a report on the use of slices of autogenous bone from the crest of the ilium in the treatment of delayed or non-union of fractures of long bones. The method has been used exclusively in this Orthopaedic Service for the past twelve years. To facilitate comparison, fractures of the tibia only have been selected for study. In the twelve years covered by this series twenty-nine tibial fractures have been grafted and the results are here reviewed.

MATERIAL

The average age of patients in this series was thirty-seven, the youngest patient being seventeen and the oldest sixty-five. The sites of the fractures were: the lowest third in eight, the junction of middle and lowest thirds in six, the middle third in ten and the uppermost third in one. Three fractures were double and one was triple. Seventeen were compound and fifteen comminuted. The initial treatment was conservative in nineteen, operative in seven and unknown in three.

Interval between injury and grafting—The longest interval between injury and grafting was ten years, the shortest eleven weeks (Table I). Four patients wearing calipers were referred from other hospitals and in these an average of four and a half years had elapsed between the time of injury and that of grafting. The longest interval in the patients treated from the outset in this centre was fifty-one weeks. Excluding the referred cases the average time between injury and grafting was twenty-eight weeks. In the clean cases the average time between injury and grafting was twenty-six weeks and in the infected ones the average period was twenty-four weeks.

TECHNIQUE OF OPERATION

A straight incision seven inches long centred on the fracture site is made over the subcutaneous border of the tibia. This is deepened directly to the bone, care being taken to avoid undercutting. With a sharp chisel the periosteum together with flakes or shingles of underlying cortical bone is raised on the lateral and subcutaneous surfaces of the tibia. At this stage the wound presents two deep gutters, each lined with osteoperiosteal flaps. It is important now to sever the periosteum in the depths of the wound on the postero-medial and postero-lateral borders of the tibia (Fig. 1). This is facilitated by the use of a chisel as a lever against the tibial shaft. Only if this is thoroughly done will it be possible to mobilise the thick edges of the wound to allow closure of the skin after insertion of the graft. Long thin slices of iliac bone approximately two millimetres in thickness are then fitted snugly with interdigitation on the tibia, between the exposed bone and the raised shingles of osteoperiosteum (Fig. 2). The wound is closed in one layer with interrupted vertical mattress sutures. A long-leg padded plaster is applied and split.

Variations in technique—In infected cases the bone is approached as far as possible through normal tissues. The graft is placed on the side of the tibia away from the infected area. The presence of a sinus is not a contra-indication to grafting but the graft should if possible be placed on the side of the tibia remote from the sinus.

Overhanging edges of tibia from lateral displacement at the fracture site are trimmed down if it seems that the deformity will be noticeable.

When mobility is excessive and a pseudarthrosis is well established a plate may be used to stabilise the fracture.

TABLE I

INTERVALS BETWEEN INJURY, OPERATION AND UNION

CLEAN CASES			
Case number	Time interval		
	Injury/Graft (*weeks*)	Graft/Union (*weeks*)	Injury/Union (*months*)
1	25	12	9½
2	24	12	9
3	20	38	14½
*4	50	12	13
5	13	16	6
6	51	15	16½
7	11 } bony union not achieved		
	72 }		
8	21	12	8
*9	312	12	81
10	56	12	17
11	21	32	13
12	13	11	6
13	17	18	9
14	21	17	9½
*15	520	12	123
16	23	12	9
17	22	12	8
18	14	12	6½
19	17	23	10
20	14	12	6½
21	42	12	13½

Average time interval: Graft/Union 15 weeks (excluding failure in Case 7)
Injury/Graft 26 weeks (excluding referred patients)
Injury/Union 11 months (excluding referred patients)

* Patients referred from other centres.

INFECTED CASES			
Case number	Time interval		
	Injury/Graft (*weeks*)	Graft/Union (*weeks*)	Injury/Union (*months*)
22	22	16	9½
23	26	—	—
	79	91	27
*24	228	24	56
25	16	12	7
26	17	24	10
27	35	17	12
28	24	60	20
29	29	10	10

Average time interval: Graft/Union 32 weeks
Injury/Graft 24 weeks (excluding referred patient)
Injury/Union 14 months (excluding referred patient)

* Patient referred from another centre.

Post-operative treatment—The first plaster is retained for six weeks, during which the patient is allowed up on crutches without bearing weight. A walking plaster is then applied and retained for a further six weeks. If the fracture is clinically solid when the second plaster is removed at twelve weeks, the patient is allowed full weight bearing out of plaster. Radiological evidence of consolidation is not expected at this stage. If the fracture is slightly mobile, further plaster fixation is indicated until clinical tests indicate that union is present. Only occasionally, in exceptionally difficult cases, is a walking caliper necessary. In such cases recovery will usually be hastened if a second graft is performed instead of temporising by the use of a caliper.

RESULTS

Union, proved radiologically, occurred after one grafting operation in twenty-seven of the twenty-nine tibiae. Two patients had two operations; in one of these union was achieved after the second graft; in the other, union failed even after the second graft and the patient elected to continue his work in a caliper rather than have an amputation which on economic grounds was the only alternative.

FIG. 1

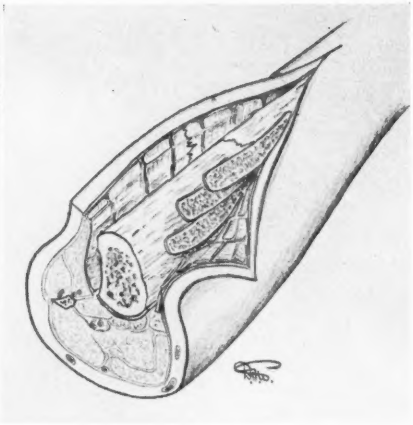


FIG. 2

Technique of operation. Figure 1—Severing the periosteum in the depths of the wound. Figure 2—The slices of bone fitted between the bone and the osteoperiosteal shingles.

Interval between grafting and union—Union was considered to be present when the fracture was sound to manual tests. In no case where clinical union was considered to be present did the fracture become mobile after unsupported weight bearing was permitted. Clinical union was present on an average twenty weeks after operation. In clean cases this interval averaged fifteen weeks and in septic cases thirty-two weeks (Table I).

Interval between injury and union—The average interval between injury and union, if the referred patients be excluded, was twelve months (Table I). The interval was eleven months in clean and fourteen months in septic cases.

The failures—In two patients the first graft was unsuccessful. The patient whose fracture did not unite after the first graft, but did after the second, presented with a severe compound fracture with loss of one inch of the tibial shaft. The patient in whom both first and second grafts failed had fractures at two levels in the tibia, with impairment of the blood supply of the middle fragment, possibly aggravated by primary internal fixation. At operation eleven weeks after injury the fracture sites were found to be mobile and the middle fragment ischaemic.

At the second operation seventy-two weeks after injury the middle fragment was a frank sequestrum and the lower fracture site was still very mobile. There was never any infection. Four years after the second operation the patient was at work wearing a below-knee caliper. The large sequestrum may have been the primary cause of failure in this case.

TABLE II

RESULTS IN TIBIAE GRAFTED WITHIN TWENTY WEEKS OF INJURY

Case number	Time interval		
	Injury/Graft (*weeks*)	Graft/Union (*weeks*)	Injury/Union (*months*)
3	20	38	14½
5	13	16	6
12	13	11	6
13	17	18	9
18	14	12	6½
19	17	23	10
20	14	12	6½
*25	16	12	7
*26	17	24	10

Average time interval: Injury/Graft 16 weeks; Graft/Union 18 weeks; Injury/Union 8 months

* Infected.

TABLE III

ANALYSIS OF INFECTED TIBIAE

Case number	Type of fracture	Infection			Skin healing			Bony union. Time after injury (*months*)
		Primary	Present at time of graft	Present after graft	Before grafting	Before bony union	After bony union	
22	Open	−	−	+			+	9½
23	Open	+	+	+				(First operation failed)
			+	+			+	27
24	Open	+	+	+			+	56
25	Open	+	−	+	+ (2 weeks)	+		7
26	Open	+	+	+			+	10
27	Closed	−	−	+			+	12
28	Closed	−	−	+			+	20
29	Open	+	−	−	+ (12 weeks)	+		10

Average time interval: Injury/Union 14 months (excluding Case 24: patient referred from another centre)

Early operation—Nine patients in whom mobility at the fracture site was detected twelve weeks after injury had their tibiae operated on within twenty weeks of injury. Two had infection at the fracture site. The average time between injury and union was eight months (Table II).

Infection—A tibia was classed as infected if a discharging sinus was present either before or after grafting. There were eight such cases (Table III). Five patients had a discharging sinus

at the time of operation. Three patients developed infection after operation though none was evident before. In three patients the wound was completely healed at the time the plaster was removed and full weight bearing was allowed, and in five union occurred before the wound was healed. If one patient who was first seen 228 weeks after injury be excluded, the average interval between injury and union was fourteen months.

CASE REPORTS

Case 18—A man of thirty injured by a motor vehicle sustained a fracture of the tibia (Fig. 3) with a small skin perforation from projection of the bone. Initial treatment was by wound closure, reduction and application of an above-knee plaster. The position obtained was unsatisfactory and nine days later a four-holed plate was placed together with chips of iliac bone across the fracture site (Fig. 4). Twelve weeks later the fracture was still mobile. After two weeks during which the skin was prepared and the muscles and joints were exercised, the plate was removed and an iliac bone graft performed. After a further twelve weeks of plaster fixation the fracture was clinically united and the patient was allowed to bear full weight out of plaster (Fig. 5). Thirteen months after operation radiographs (Fig. 6) showed consolidation of the graft with almost complete obliteration of the fracture line.

Case 23—A man of twenty-four injured by a bus sustained a severely compound comminuted fracture of the right tibia and a comminuted fracture of the lowest third of the right femur (Fig. 7). During the initial debridement it was found that a one-inch segment of the tibial shaft had been lost. Steinmann pins were inserted through the upper end of the tibia and through the calcaneum and were fixed to the side bars of a Thomas's splint with McKee clamps. By this means it was possible to reduce the size of the gap left between the tibial fragments. Eight weeks later a hip spica was applied. It was retained for six weeks and at this time, fourteen weeks after injury, the femoral fracture was united in good position but the tibial fracture showed distinct movement. It was therefore decided to perform a bone graft, but before this could be done the wound over the tibial fracture broke down. It was still discharging slightly at operation twenty-six weeks after injury (Fig. 8). At operation the sinus was avoided and iliac slices were placed subcortically on the subcutaneous and lateral surfaces of the tibia. No infected bone was observed at operation. On removal of the plaster five months later the wound was healed but there was still a slight movement at the fracture site. A caliper was fitted and retained for seven months, during which time the wound again began to discharge. It was not completely healed twenty months after injury (Fig. 9). At that stage it was decided to perform a second grafting operation. Three months after this the fracture was clinically united but the wound continued to drain slightly and went on doing so for some months. Two years after the second operation the patient is an active outdoor worker and states he can do everything he could before the injury. Radiographs (Fig. 10) show osseous union with partial obliteration of the fracture line. There is still a small ulcer present at the site of the wound.

DISCUSSION

This method is based on the assumption that it is never necessary to freshen the ends of the bone fragments before applying a graft because the fibrocartilaginous material between them is capable of ossification; and that the biological activity which leads to union resides in the ends of the bone and not in the graft. Elmslie, as quoted by Burrows (1940), stated that he never freshened the ends of the fragments nor did he consider rigid internal fixation necessary. The sliding cortical graft which he employed was never fixed with screws. Nor did Dunn (1939) consider it necessary to use internal fixation or to freshen the bone ends. He went even further in showing that it was frequently unnecessary to use a graft if the surfaces of the bone were turned up as osteoperiosteal shingles. It was, however, the writings of Phemister (1947) which led directly to the development of the procedure described in this paper. In the technique used in this series strips of cancellous bone, used in the manner described by Phemister, have been combined with the subcortical procedure of Dunn and for this reason the method has been called by Charnley (1957) the " subcortical grafting of iliac bone."

That fresh autogenous cancellous bone forms an excellent bone graft is now generally accepted since Mowlem (1944) drew attention to it. In view of the excellence of the results of applying this type of graft for delayed union of fractures of long bones it is surprising that so

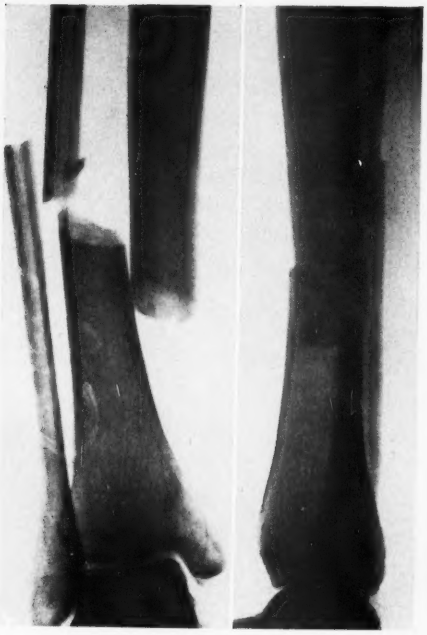


FIG. 3

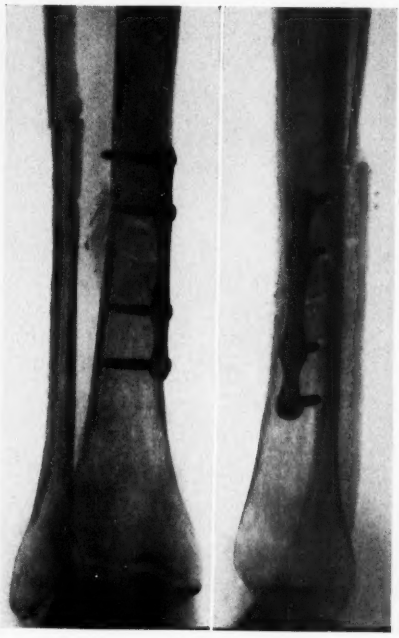


FIG. 4

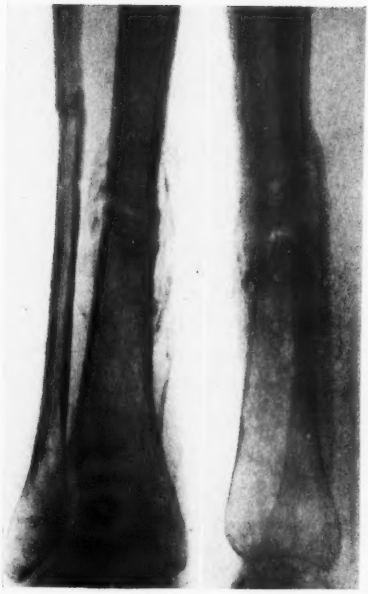


FIG. 5

FIG. 6

Illustrating progress in Case 1. Figure 3—Initial radiographs. Figure 4—Position after internal fixation. Figure 5—Twelve weeks after bone grafting. Union clinically present. Figure 6—Consolidation thirteen months after operation.

FIG. 7

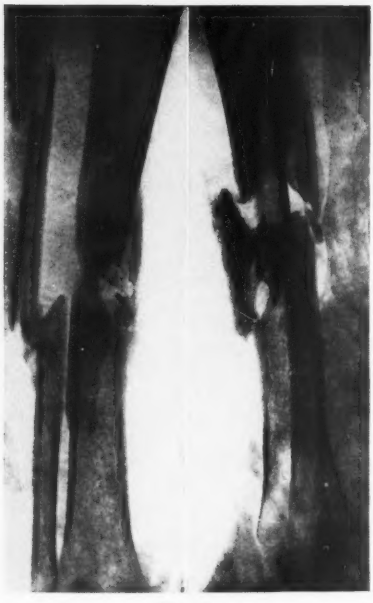


FIG. 8

FIG. 9

FIG. 10

Illustrating progress in Case 2. Figure 7—Initial radiograph. Figure 8—Twenty-six weeks after injury. Figure 9—Fourteen months after bone grafting. Failure of bony union. Figure 10—Two years after second bone graft. Bony union with partial obliteration of fracture line.

little published work dealing with a significant number of operations is available. Though there is no doubt that methods similar to the one described here are in current use by many surgeons, the only published reference to a significant series which this writer has traced is a brief résumé of a paper by Wilson (1957). The evidence presented here shows that cancellous bone grafting is not only highly effective in clean cases, but also that it can secure union despite the presence of mild sepsis. In fractures which are recently compound, or which continue to drain slightly at the time of operation, grafting can be performed with minimum risk.

It is emphasised that this technique of bone grafting is best used early in delayed union and that it is regarded as a prophylactic measure against non-union. Use in this manner should reduce the time between injury and bony union.

SUMMARY

The use of a subcortical graft of autogenous iliac bone in patients with delayed union or non-union of fractured tibiae is described together with the results in twenty-nine patients. This procedure when performed early even in the presence of mild sepsis makes it possible to shorten the time of healing without any undue risk.

I am glad to acknowledge my indebtedness to Mr John Charnley for permission to make this study from his records, radiographs and patients during the period of my appointment as Orthopaedic Registrar at Park Hospital, Davyhulme.

REFERENCES

BURROWS, H. J. (1940): Treatment of Ununited Fractures by Bone Grafting without Resection of the Bone Ends. *Proceedings of the Royal Society of Medicine* (Section of Orthopaedics), **33,** 157.

CHARNLEY, J. (1957): *The Closed Treatment of Common Fractures.* Second edition. Edinburgh and London: E. & S. Livingstone Ltd.

DUNN, N. (1939): Treatment of Ununited Fractures. *British Medical Journal,* **ii,** 221.

MOWLEM, A. R. (1944): Cancellous Chip Bone-grafts. *Lancet,* **ii,** 746.

PHEMISTER, D. B. (1947): Treatment of Ununited Fractures by Onlay Bone Grafts without Screw or Tie Fixation and without Breaking Down of the Fibrous Union. *Journal of Bone and Joint Surgery,* **29,** 946.

WILSON, J. N. (1957): Cancellous Strip Bone Grafting. *Journal of Bone and Joint Surgery,* **39–B,** 585.

FRACTURES OF THE FOREARM TREATED BY RIGID FIXATION

J. H. HICKS, BIRMINGHAM, ENGLAND

From the Birmingham Accident Hospital

This paper describes the results of exceptionally rigid fixation of fractures of the forearm by a specially designed plate. This plate provides greater measured rigidity than that afforded either by plaster immobilisation or by any other plate in common use, the control of rotation being specially good.

MATERIAL

The fractures, with two exceptions, were in patients aged from fifteen to seventy-five years. Cases were selected for their unpromising characteristics, compounding for example often being taken as an indication for use of the treatment. No type of fracture of the shaft of the bone was excluded. In only a very small number with extensive comminution did it prove impossible to apply the method.

The first fifty-one patients in the series were followed up for at least a year, when their forearms were examined for range of movement, power and general usefulness. Radio-ulnar movement was measured by Patrick's goniometer; flexion and extension of the wrist were measured by radiography; the range of elbow movement was measured by a goniometer; power of grip was measured by a dynamometer. These values were expressed as percentages of those of the opposite healthy limb. Precise measurement of the range of movement of the elbow was discontinued when it became clear that 90 per cent of the elbows regained 90 per cent or more of the normal range. Only one elbow showed significant limitation (76 per cent of normal range), caused by fibrosis of the scar and formation of a web.

THE METHOD OF INTERNAL FIXATION

The plate—The plates used in this series were made of molybdenum stainless steel (18/8 Mo or En 58 J). Plates made of cobalt-chrome alloy are now available. Their specification is as follows (Fig. 1): length 3 inches; width, not less than 0·275 inch; thickness, not less than 0·118 inch; radius of curvature $\frac{9}{32}$ inch; outside diameter of screws accepted by the holes $\frac{7}{64}$ inch approximately; inclination of screws to each other 60 degrees; bone drill sizes (both required) $\frac{3}{32}$ inch for cortical bone, and number 44 (alternatively $\frac{5}{64}$ inch) for cancellous bone.

Maximal rigidity has been achieved by using the largest possible cross-section, thickness being more important than width. Rigidity is also preserved by the absence of screw-holes in the main structure of the plate except at the ends where they cannot weaken it. Control of rotation is achieved by offsetting four of the screw-holes in lugs $\frac{5}{32}$ inch on either side of the mid-longitudinal line. This results in the screws entering the shaft at circumferential points separated from each other by about one-sixth of the circumference of the bone. With this arrangement it is impossible for either fragment to rotate on the plate without one or other of the screws being pulled perpendicularly out (Fig. 2).

The screws—The screws are $\frac{1}{32}$ inch smaller than the screws in common use such as the standard cobalt-chrome screw. They were chosen to reduce the risk of damaging bone by the drilling of large holes through it. The standard screw, which occupies up to one-sixth of the diameter of the radius, seems to introduce an unjustifiable risk to blood supply and hence to union. Necrosis of the end of a fracture fragment beyond the last screw-hole has in the past occasionally been seen. Another risk of using the larger screws is fracture from later injury across one of the large holes made by them.

Technique of fixation—The plates are more difficult to apply than conventional ones because they have to conform to the bone in three dimensions.

Approach—For the radius the approach described by Henry (1957), but usually omitting the division of brachioradialis, is most generally useful. For the distal half of the bone an approach posterior to brachioradialis gives access to the more easily plated aspect. In either case the radial nerve must be seen, firstly because it affords a valuable landmark distinguishing brachioradialis and extensor carpi radialis longus and secondly because division causes a patch of numbness that troubles the patient. Ample length of incision is required because of the wide angle of approach needed by the drill and screwdriver for the divergent screws. With the radius, especially the deep proximal half, the need for heavy retraction is avoided by rotating the bone to bring each screw-hole in turn to face the operator.

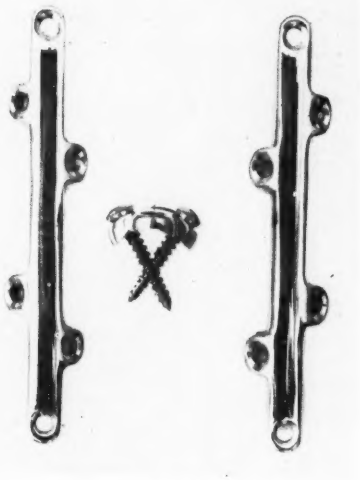


FIG. 1

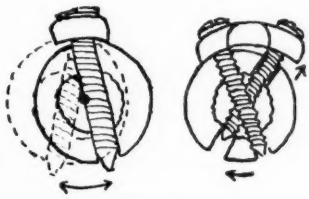


FIG. 2

FIGS. 1 AND 2

Figure 1—The lug-plate. Note the two patterns, to suit obliquity of the fracture in either direction, and the position and direction of the screws in the end-on view. Figure 2—The protection against rotation of the bone on the plate given by the lug-plate (*right*) compared with that given by the standard plate (*left*).

Shaping and fitting—The plate should be applied over the periosteum. It is possible—and often essential—during operation to bend the plate in its longitudinal axis to fit the curvature of the bone. It is not possible to alter the curvature of its cross-section and, since the curvature of the cross-section of the bone varies at different places, some trial has to be made to achieve the best fit. In the lower half of the radius the best sites are the lateral or dorsilateral surfaces. Sometimes the plate cannot be made to fit the ulna and a conventional plate has to be used. Care has to be taken to see that all the lugs are lying snugly against the surface before the drill-holes are started or a forced rotational deformity will be imposed at the fracture site. In practice slight rotational deformity or distraction has not delayed union.

Drilling and screwing—A small screwdriver is preferable because it is possible to break the screws if much force is used on them. Of the two drills recommended, the larger ($\frac{3}{32}$ inch) is used to make the holes through cortical bone—with the smaller size the screws may jam and break. One or other of the smaller drills is used to make the hole (usually only the lowest one) through cancellous bone—a larger hole might cause stripping of the bony thread during insertion of the screw. A little extra care is necessary to avoid breaking these small drills. Every screw-hole must, of course, be used and every screw should have a hold on both cortices. The length of screw required is generally three quarters of an inch but with a fracture

low in the radius the widening of the lower end of the bone will necessitate a length of the lowest screw of up to one and a quarter inches. Very occasionally where comminution is extensive the central shank of the plate may prove to be too short to allow it to be used.

TABLE I

RELIABILITY OF UNION

(The two patients under fifteen years old have been excluded because these fractures in children present no problems of union)

Type of fracture	Number of forearms	Number compound	Number united without other major treatment
Fracture of radius with inferior radio-ulnar subluxation or dislocation . . .	24	1	24
Solitary fracture of radius . . .	1	0	1
Solitary fracture of ulna	9	0	9
Fracture of both forearm bones . .	25 (32)	9	21 (28)
Totals	59 (66)	10	55 (62)

(Figures in brackets represent numbers of *bones* treated by rigid internal fixation.)

TABLE II

TIME TO CONSOLIDATION

	Number of bones plated
First three months	4
Second three months . . .	27
Third three months	15
Fourth three months . . .	14
Fifth three months	2
Non-union (requiring some other major treatment before union occurred) .	4
Total	66

TABLE III

FUNCTION AT ONE YEAR

Number of forearms	Number showing range of pronation-supination of 80 per cent or more	Number showing range of flexion-extension of wrist of 80 per cent or more	Number showing power of grip of 80 per cent or more
51	38 (74 per cent)	41 (80 per cent)	31 (61 per cent)

After-treatment—The fixation is usually strong enough to hold without any external splinta ge but a lot depends upon what sort of regime is followed. Before bony union has occurred t ie fixation will not stand up for certain to the stresses imposed by manual work or those impos d

by physiotherapy no matter whether the limb be in or out of plaster. In the case of the patient with concomitant injuries to the lower limb it is unwise to expose the fixation to the strain imposed by the use of crutches. Two of the four instances of non-union may have been produced in this way. A limb in which a broken bone has been rigidly plated is from the early days very free from pain and stiffness and both surgeon and patient may be tempted to take risks. It is difficult to judge when union has occurred and easy to be misled by the literature into thinking that it takes less time than it actually does. Mechanical breakdown is always heralded by pain but if precautions to reduce the forces are immediately taken a catastrophe may even then be averted. Prevention, however, would have been preferable.

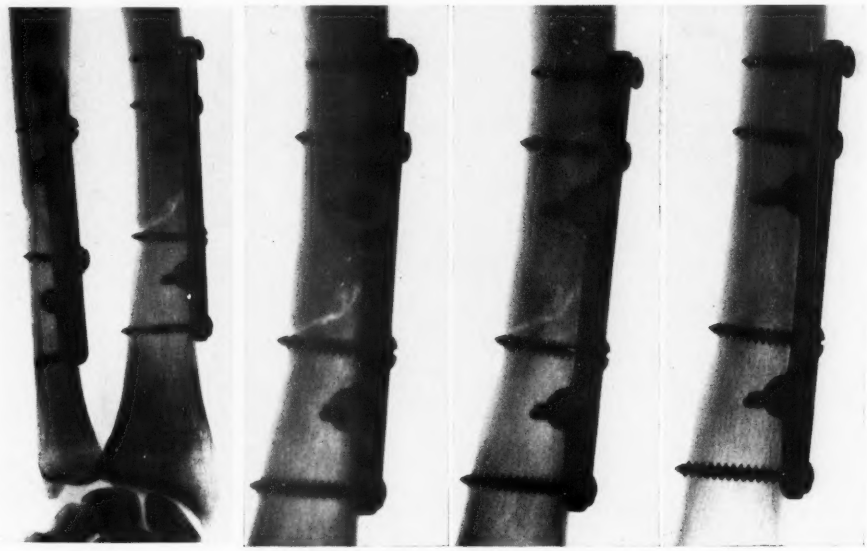


FIG. 3 FIG. 4 FIG. 5 FIG. 6

Union with minimal callus when rigid fixation is used. Figure 3—Compound fracture of radius and ulna ten weeks after fixation. Figure 4—The radial fracture at four months. At this stage the patient went back to work. Figure 5—The radial fracture at six months, showing gradual obliteration of the fracture line. No visible external callus. Figure 6—The radial fracture at one year. Consolidation now considered to be present.

RESULTS

Reliability of union—Nearly ninety of the special plates have now been used and final results from the first sixty-six are given in Table I. Non-union occurred in four out of fifty-nine forearms (7 per cent) or in four out of sixty-six broken bones (6 per cent).

Time to consolidation—Because of the difficulty in determining exactly when union has occurred the " time for consolidation " is given (Table II) in preference to the " time to union." This stage is reached approximately two or three months after union (Figs. 3 to 6). Most of the patients were back at work long before this stage; a number were back at work (some safely but many ill-advisedly) even before union. One man was playing the piano professionally and one woman was doing light housework for a month or two before radiological union. An over-enthusiastic housewife even did the washing-up—cautiously—on the evening of the day of operation. Ability to do light work, therefore, although an asset, is certainly not a sign of union since with rigid fixation the strain can be taken by the plate. Several patients who were tempted or permitted to do heavy work at this stage came near to catastrophe.

Union with minimal callus—The difficulty referred to above in judging when union has occurred radiologically arises in any series of rigidly fixed fractures because of a tendency to develop

FIG. 7

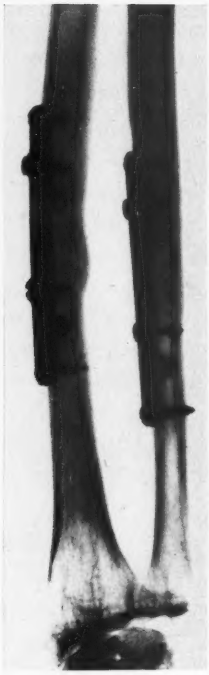


FIG. 8

FIG. 9

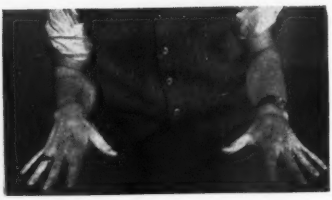


FIG. 10

A below-average result from lug-plating. Figure 7—Antero-posterior (*left*) and lateral (*right*) radiographs showing the fractures. Figure 8—Consolidation after fixation by lug-plates. Figures 9 and 10—The functional result, showing range of movement of wrist and radio-ulnar joints.

little or no external callus (Figs. 3 to 6). This peculiar behaviour has been described previously (Danis 1949, Blockey 1956, Hicks 1959).

Final functional results—After tabulation of the three most important measurable functions (pronation-supination and flexion-extension of the wrist and power of grip) a line was drawn separating those patients with 80 per cent or more of these functions from the rest (Table III). Clinical assessment shows that this line of separation divides those with thoroughly satisfactory function from those who noticed some deficiency in ordinary everyday use. An example of this is given in the following case report, which has been selected to show the poorest result in the " satisfactory " group.

Case report—A young man broke his left forearm in a motor-cycle accident (Fig. 7). Both bones were lug-plated (Fig. 8). No plaster splint was used at any time. The patient returned to work as a draughtsman three months after operation. (In this he took a risk because neither bone was united.) Considerably before the one-year assessment (Figs. 9 and 10) he was using the limb for all its pre-accident activities and he could detect only slight limitation of supination and some loss of power—a fractured metacarpal being probably responsible for some of the latter. He had resumed his motor-cycling and was doing the repairs to and maintenance of his own machine. The one-year assessment (Figs. 9 and 10) was as follows: wrist range 85 per cent; pronation-supination range 86 per cent; elbow range full; power of grip 70 per cent.

COMPLICATIONS

Infection—Eighty-one of eighty-two forearms healed quickly with no trouble (Table IV). There was only one instance of infection, and that was in a severely compound fracture. It was a staphylococcal cellulitis occurring during the first week after operation. Treatment was considered unnecessary and the infection cleared up completely by the end of the second week. It cannot therefore be regarded as serious.

TABLE IV

INFECTION

(This Table includes more recent cases)

Conditions in which the lug-plate was used	Number of operations	Cases of infection
Simple fractures or late treatment . .	74	0
Immediate treatment of compound fractures	8	1

Micro-corrosion—Fifteen per cent of plates have had to be removed because they caused mild symptoms of corrosion. Now that the plates and screws are available in cobalt-chrome alloy a much lower incidence of this trouble can be expected.

DISCUSSION

Reliability of union—Rates of non-union have been calculated from data supplied in the five most important recent papers. The results obtained in the present series are found to show a substantial improvement (Table V). Furthermore the 3 per cent bone-graft rate in the present series compares favourably with the 18 per cent reported by Cowie (1956). In only one paper (Ritchey, Richardson and Thompson 1958) are results claimed that are better than those of the present series. However, their criteria for union are not very strict since one of the illustrations shows a fracture in a serviceman two weeks before " discharge to duty " in which

one, and possibly both, bones are by our standards completely ununited. As mentioned above, ability to return to light work is not to be regarded as proof that union has occurred or even that future progress will be straightforward.

The comparison with Smith's (1959) work is of particular significance because the work reported in both papers comes from the same hospital. There was the same " watershed," almost the same group of surgeons and access to identical ancillary services. Smith's series

TABLE V

INCIDENCE OF NON-UNION

		Incidence of non-union (per cent)	Remarks
All types of forearm fracture	Knight and Purvis (1949)	15	
	Robertson (1953) . .	20	Treatment by medullary fixation
	Smith and Sage (1957) .	14	Treatment by medullary fixation
	Smith (1959) . .	17	
	The present series . .	7	
Fractured radius with inferior radio-ulnar subluxation or dislocation	Hughston (1957) . .	17	
	The present series . .	nil	Twenty-four consecutive cases

terminated and this series started in 1956. The improvement cannot be attributed to Smith's discovery that late plating was giving more reliable union than early plating, because investigation shows that this advice has not been acted upon and that the proportion of early platings remains unchanged. The only new factor has been the introduction of the lug-plate. It seems that whereas Smith's rule is valid when applied to the results of ordinary plating it becomes less important in the case of rigid plating.

Severity of the fractures—The only way of comparing the severity of the various series is by comparing the proportion of compound fractures. Robertson's (1953) series had 30 per cent, Cowie's (1956) 22 per cent, Smith and Sage's (1957) " one-third," and Smith's (1959) 15 per cent. In the present series there were eleven compound in sixty-two fractured forearms (18 per cent).

Infection—The incidence of infection in the present series, one in eighty-two, compares favourably with the twenty-seven in 338 (one in thirteen) of Smith and Sage (1957) but may be poorer than the one in 102 found by Smith (1959). There were no cases of infection in the fifty-four reported by Cowie (1956). Our one infection occurred in a compound fracture and it has been shown elsewhere (Hicks 1957) that much of the infection in plated compound fractures is due to the compounding and not to the plating.

Function—Although restoration of function is rightly referred to as the true objective of treatment, no attempt at accurate quantitative assessment can be found in the literature.

Robertson (1953) claimed that, if cases of non-union were excluded, forearm rotation in his series was never less than 90 degrees. Amongst corresponding cases in the present series there were two with a rotation limited to 35 degrees because of cross union. In each of these there had been a mixture of treatments—no internal fixation for one bone and rigid fixation for the other—and it is therefore impossible to attribute the poor result to any one treatment. None of the other patients in the series had a range of rotation of less than 100 degrees while most had a much greater range (average 165 degrees).

Seventy-two per cent of the patients reported by Cowie (1956) had a final range of rotation that may have been 75 per cent or over—the accuracy of the measurements is uncertain. In the present series 74 per cent had a range of 80 per cent or over.

Causes of failure and near-failure—In addition to the cases of non-union there were several in which success was certainly not attributable to any virtue of the plate. Apart from errors of technique most of the failures and near-failures were due to forces that were too large being applied too soon, in other words, a return to everyday use when union was absent or only present at an early stage. The temptation to hurry things is strong because of the confidence inspired in both surgeon and patient by the painless free movement which from the start is characteristic of rigid fixation. The difficulty of judging is made greater by the lack of reliable radiological criteria of union. Pain whenever it occurs ought to be regarded as a warning that movement is occurring at the fracture site. In upgrading activity it is better to be a month or two too late than a day too soon.

SUMMARY

1. A series of fractures of the forearm has been treated by exceptionally rigid internal fixation with a special plate and screws.
2. The plate and screws are described.
3. The results of rigid fixation are found to be: i) reliability of union, and ii) good final function.
4. The lessons learned regarding the application of the plate and the after-treatment of the forearms are recounted.

Acknowledgments are due to my colleagues at the Birmingham Accident Hospital for permission to quote their cases.

REFERENCES

BLOCKEY, N. J. (1956): The Value of Rigid Fixation in the Treatment of Fractures of the Adult Tibial Shaft. *Journal of Bone and Joint Surgery*, **38–B,** 518.

COWIE, R. S. (1956): Fractures of the Forearm Treated by Open Reduction and Plating. *British Journal of Surgery*, **44,** 263.

DANIS, R. (1949): *Théorie et pratique de l'osteosynthése.* Paris: Masson et Cie.

HENRY, A. K. (1957): *Extensile Exposure.* Second edition. Edinburgh and London: E. & S. Livingstone Ltd.

HICKS, J. H. (1957): The Relationship between Metal and Infection. *Proceedings of the Royal Society of Medicine* (Section of Orthopaedics), **50,** 842.

HICKS, J. H. (1959): In *Modern Trends in Accident Surgery and Medicine.* Edited by Clarke, R., Badger, F. G., and Sevitt, S. London: Butterworth & Co. (Publishers) Ltd.

HUGHSTON, J. C. (1957): Fracture of the Distal Radial Shaft. *Journal of Bone and Joint Surgery*, **39–A,** 249.

KNIGHT, R. A., and PURVIS, G. D. (1949): Fractures of Both Bones of the Forearm in Adults. *Journal of Bone and Joint Surgery*, **31–A,** 755.

RITCHEY, S. J., RICHARDSON, J. P., and THOMPSON, M. S. (1958): Rigid Medullary Fixation of Forearm Fractures. *Southern Medical Journal*, **51,** 852.

ROBERTSON, R. C. (1953): Intramedullary Fixation of Fractures of the Forearm. *American Journal of Surgery*, **85,** 496.

SMITH, H., and SAGE, F. P. (1957): Medullary Fixation of Forearm Fractures. *Journal of Bone and Joint Surgery*, **39–A,** 91.

SMITH, J. E. M. (1959): Internal Fixation in the Treatment of Fractures of the Shafts of the Radius and Ulna in Adults. *Journal of Bone and Joint Surgery*, **41–B,** 122.

AVASCULAR NECROSIS AFTER SLIPPING OF THE UPPER FEMORAL EPIPHYSIS

H. G. LOWE, STAFFORD, ENGLAND

Necrosis in the femoral head after slipping of the upper femoral epiphysis has always been regarded as serious. This paper is a study of hips with slipped upper femoral epiphyses that showed evidence of necrosis either when first seen or during treatment. The objects were to find out whether the necrosis affected bone or articular cartilage, or both, whether there was any common factor in the patients in whom necrosis occurred, and to what extent treatment influenced the ultimate state of the hip.

REVIEW OF LITERATURE

The reported incidence of avascular necrosis after slipping of the upper femoral epiphysis treated by different methods has varied. Details of patients showing necrosis are not often mentioned, and few radiographic findings have been shown in the reports. As Jerre (1950) observed, many authors have not distinguished between avascular necrosis of the femoral head and early necrosis of articular cartilage unaccompanied by any radiological signs of necrosis of bone; nor has a distinction always been made between early necrosis of articular cartilage and secondary degenerative arthritis.

The term " aseptic " necrosis of bone appears to have been used first by Axhausen (1922), who described it in the femoral head after fracture of the femoral neck in a child; he also thought that similar changes occurred after slipping of the upper femoral epiphysis. Waldenström (1930) recognised the existence of a type of joint change after slipping of the upper femoral epiphysis which involved necrosis of the articular cartilage without radiological evidence of avascular necrosis of the capital epiphysis. His paper described three such cases that he had seen in 1923. Jones and Roberts (1934) clarified the radiological diagnosis of avascular necrosis of bone in terms of contrasting bone densities caused by different blood supplies; the area of bone that is deprived of its blood supply retains its calcium salts and hence its density on radiography, in contrast to the surrounding bone which retains its normal blood supply and undergoes disuse demineralisation with calcium absorption and which therefore appears rarefied in radiographs.

Moore (1945*a*) gave the first detailed description of the pathology of avascular necrosis in the femoral epiphysis after slipping, from a careful study of two specimens obtained at operation. He also reported one patient with necrosis of the articular cartilage of both hips in a series of forty-four slipped upper femoral epiphyses. Ponseti and Barta (1948) reported three patients with necrosis of the articular cartilage after slipping of the upper femoral epiphysis, and Jerre (1950), in a series of 200 slipped femoral epiphyses—treated mainly by closed reduction and plaster immobilisation—found twenty hips with avascular necrosis and nine with necrosis of the articular cartilage. Hall (1957) reviewed 173 hips with slipping of the femoral epiphysis from various orthopaedic centres; twenty-seven of these showed avascular necrosis of the capital epiphysis, and three had necrosis of the articular cartilage.

CLINICAL MATERIAL

The material on which this paper is based is drawn from a series of 100 cases of slipped upper femoral epiphysis treated at the Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry, and at the Prince of Wales Orthopaedic Hospital, Cardiff, in twenty-five years. Of these, twenty-one hips in twenty patients developed necrosis of the bony epiphysis (Table I

or the articular cartilage (Table II). The diagnosis of avascular necrosis of bone was made on the radiological finding of increased density of the epiphysis in six hips. The diagnosis of articular cartilage necrosis—based on the radiological finding of diminished joint space and the clinical findings—was made in fifteen hips and confirmed at operation in three. In some patients the changes of articular cartilage necrosis were diagnosed when the patient was first examined. In others—and in all with bone necrosis—the changes developed within the first year. Figures 1 and 2 show examples of the two types of necrosis.

FIG. 1

FIG. 2

The two types of necrosis after slipping of the upper femoral epiphysis. Figure 1 (Case 8) shows necrosis of the articular cartilage with loss of joint space and porosity of the head. Figure 2 shows necrosis of the upper half of the bony epiphysis, but with a good joint space (Case 3).

The small number of hips with avascular necrosis of the epiphysis is in contrast to that in other published series. Perhaps this can be explained in part by the failure of some authors to distinguish between the two types of necrosis. It is also possible that some bone necrosis may have been missed in this series by infrequent radiography, but this is unlikely because the radiographic changes of altered density usually become apparent in the early months, and most of these patients were in hospital at that time undergoing radiographic examination at monthly intervals.

The two forms of necrosis will be considered separately because, although they cannot be separated into two distinct pathological types, they appear as separate clinical entities with different radiological appearances.

AVASCULAR NECROSIS OF THE CAPITAL EPIPHYSIS

There seems general agreement that if the blood supply to the capital epiphysis is impaired, partial or total death will occur in the tissues of the epiphysis depending on how much the blood supply is diminished. Waldenström (1934) separated the capital epiphysis from its connections with the femoral neck and the ligamentum teres, and then replaced it on the neck and held it there by means of a bone peg hoping that it would unite as a free bone graft. He did this twice but avascular necrosis of the head occurred in both. Zemansky and Lippmann (1929) cut the artery of the ligamentum teres in adolescent rabbits and found that this caused anaemic bone necrosis in the femoral head. Harris and Hobson (1956) showed, also in rabbits, that necrosis followed experimental displacement of the upper femoral epiphysis.

TABLE I

CLINICAL DETAILS IN SIX PATIENTS (SIX HIPS) WITH AVASCULAR NECROSIS OF THE FEMORAL HEAD AFTER SLIPPING OF THE EPIPHYSIS

Case number	Sex	Age in years	Avascular necrosis		Result
			Onset	Duration	
1	M	15	At 3 weeks	3 months	Good 1 year and 16 years later
2	M	14	At 5 weeks	13 months	Good 1½ years and 9 years later
3	M	16	At 6 weeks	5 months	Good function 5 years later
4	F	9	Before 3 months	2 years	Good 15 years later
5	M	16	Before 5 months	1 year	Good function 2 years later
6	F	13	At 4 months	—	Density of head improving 14 months later

TABLE II

DETAILS OF FOURTEEN PATIENTS (FIFTEEN HIPS) WITH NECROSIS OF CARTILAGE AFTER SLIPPING OF THE UPPER FEMORAL EPIPHYSIS

Case number	Sex	Age in years	Early findings		Late findings		Results
			Joint space	Movements	Joint space	Movements	
7	M	16	Reduced	Very limited	Very narrow	Limited	Very limited movements. Flexion contracture
8	M	15	Reduced	Slightly limited	Practically none	None	Arthrodesed
9	M	16	Normal	Restricted by pain	—	Fibrous ankylosis	Arthrodesed
10	M	18	Reduced	Very limited	Diminished	Limited	Intertrochanteric osteotomy at 9 months. Final result moderate
11	M	16	Normal	Restricted by pain	Progressive loss	Very limited	Arthrodesed
12	F	13	Normal	Limited	Very diminished	Very poor	Awaiting osteotomy
13	F	11	Right: Normal	—	Very diminished	Very poor	Abduction deformity
			Left: Normal	—	Very diminished	Very poor	—
14	M	14	Normal	Limited by spasm	Diminished	Quite good	—
15	F	13	Good	Limited by pain	Diminished at 2 years	Poor at 12 years	Secondary arthritis 12 years later
16	M	20	Normal	Very limited	Arthritis at 2 years	Very poor at 2 years	Painful hip
17	M	15	Slight loss	Some limitation	Poor	Limited	—
18	M	14	Normal	—	Diminished at 1 year	Poor at 12 years	Osteotomy at 4 months. Arthritis at 12 years
19	M	15	Normal	Restricted	Good at 15 months	Good at 15 months	Loss of joint space and restriction of movement 2 years later after immobilisation of opposite hip
20	F	15	Greatly reduced	None	—	—	Spontaneous fibrous ankylosis

The anatomical vascular pattern in man has been studied by dye injection and by naked-eye dissections of anatomical specimens, and opinion has differed as to the relative importance of the various vascular sources, particularly at different ages. Trueta (1957) supported the view that the lateral epiphysial vessels and the foveal vessels in the ligamentum teres form the main blood supply to the femoral head during adolescence.

Some authors have observed that avascular necrosis can be caused by displacement of the epiphysis alone, without treatment. Moore (1945*b*) stated that "epiphysial necrosis may occur naturally with minimal displacement." In the present series no patient showed evidence of sclerosis of the head when first examined radiologically; all the patients received some form of treatment before the sclerosis appeared and, although in this paper it has been assumed that treatment was responsible for the necrosis, the possibility that it was a direct sequel of displacement cannot be disproved.

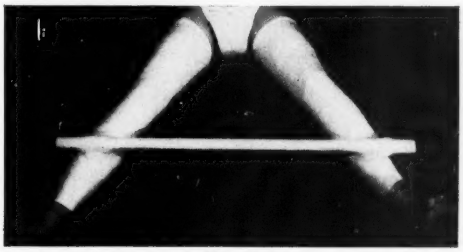


FIG. 3
Anti-rotation plasters allow flexion and extension but prevent rotation.

All the six patients with avascular necrosis of the femoral head had considerable displacement when first seen, with a normal joint space and bone density, and with symptoms of less than four weeks' duration which were mostly attributed to a recent fall. In four patients movements in the affected hip were restricted at first by pain and spasm.

The site of the sclerosis in the femoral head varied. In one patient treated by manipulation and nailing the whole head became dense. The upper half of the head became avascular in three patients, two of whom were treated by manipulation and nailing, and the other by open reduction and nailing. In two patients the necrosis occurred in a central area. One of these patients was treated by medial rotation plasters after traction (Fig. 3) and the other was treated by traction, during which the head was distracted from the acetabulum, and this probably occluded the vessels in the ligamentum teres and led to the central necrosis. The three patients who showed increased density of the upper half of the femoral epiphysis had probably suffered rupture of the lateral epiphysial vessels while retaining the vessels in the ligamentum teres. The patient in whom the whole head became dense either must have lost both sources of blood supply or had an underdeveloped vessel in the ligamentum teres: this occurs in some people and makes the epiphysis particularly vulnerable.

In all the hips the position of the epiphysis was improved and in two the displacement had been slightly overcorrected into the valgus position.

The findings in this small series agree with those of Jerre (1950), who found that a successful manipulative reduction was more likely to cause avascular necrosis than an unsuccessful manipulation, and that severe epiphysial displacements after injury showed a relatively high incidence of avascular necrosis.

The necrosis in three patients showed within six weeks; this is rather earlier than is usually reported. Two of the six patients recovered normal bone density within four months; both had started non-weight-bearing exercises immediately after the insertion of a Smith-Petersen nail. This contrasts with three other patients who took over twelve months to regain normal density and who had not been treated by immediate mobilisation. This gives some support to the view expressed by Watson-Jones (1943) that "revascularisation is promoted best by active non-weight-bearing exercise."

The fate of the articular cartilage after necrosis of the bony epiphysis probably influences the outlook for joint function as much as the amount of bony deformity resulting from collapse of necrotic bony tissue. In the experimental work on rabbits by Harris and Hobson

(1956), separation of the capital epiphysis caused necrosis of the bone; the articular cartilage showed cellular changes and the intercellular substance became fibrillated. The number of pathological reports on human specimens in the literature is small, and the two described by Moore (1945*a*) are the only ones showing in microscopic detail the bone and cartilage changes in an upper femoral epiphysis that had developed avascular necrosis from slipping. In the first, a boy aged twelve, the bone of the epiphysis underwent necrosis but most of the articular cartilage appeared alive on section, although a little thinner than normal. The second specimen

FIG. 4

FIG. 5

FIG. 6

FIG. 7

FIGS. 4 TO 7

Case 1—A boy of fifteen was found to have a slipped upper femoral epiphysis with a history of two weeks (Fig. 4). The femoral head became dense, but this improved with mobilisation after pinning. Sixteen years later the hip was satisfactory with a good range of movement (Figs. 5 to 7).

was obtained from a man aged twenty-one; there was non-union between the necrotic epiphysis and the metaphysis; most of the articular cartilage over the dead bone had become necrotic, but one area of cartilage survived. The changes found in both specimens were late findings. There was a considerable difference in age between the two patients, and the older one had no evidence of the development of secondary sexual changes. The fact that most of the articular cartilage survived in the specimen from the younger boy is significant, as Moore suggests, and recalls the changes found in Perthes' disease in which the articular cartilage

survives well, and is indeed sometimes hypertrophied. Patients with Perthes' disease are admittedly younger, but it is probable that in adolescents the nutrition of the articular cartilage resembles that in younger children. That the cartilage obtains its nutrition from the synovial fluid rather than directly from the underlying blood vessels in the subchondral layer is supported by physiological and pathological studies (Bauer, Ropes and Waine 1940; Landells 1957). Landells believed that direct contact of blood vessels with the articular cartilage causes death of the cartilage cells. If it is true that the nourishment of the articular cartilage is derived

FIG. 8

FIG. 9

FIG. 10

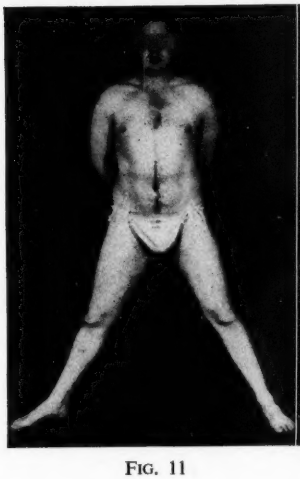


FIG. 11

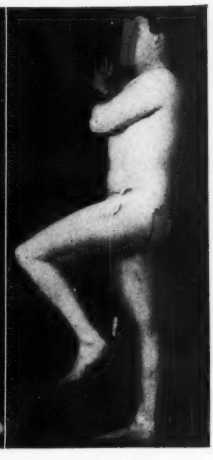


FIG. 12

FIGS. 8 TO 12

Case 2—A boy of fourteen. Figure 8 shows the appearance of the hip on admission. Nine weeks after manipulation and nailing the head is dense (Fig. 9) but nine years later the hip is satisfactory (Fig. 10) with a good range of movement (Figs. 11 and 12).

from the synovial fluid, then the onset of bone necrosis would not be expected to cause complete death of the articular cartilage provided the synovial membrane and vessels in the joint capsule carried on their normal physiological role. This was shown to be the late result in four of the patients in this series (Figs. 4 to 12, Cases 1 and 2), each patient having a reasonable joint space, a good range of movement and a fairly well shaped head.

In this series it is shown that avascular necrosis can follow a successful manipulation of a recent severe epiphysial slip, but that the prognosis is not necessarily bad because the

articular cartilage can survive, especially if aided by early mobilisation. Osteoarthritis may develop later, but at least there can be several years of good painless function. This prognosis is very different from that of necrosis of articular cartilage, which is extremely poor.

NECROSIS OF ARTICULAR CARTILAGE

Since Waldenström first described necrosis of the articular cartilage in three hips in 1930, sixteen other cases have been recorded in the literature. In this series there was necrosis of the articular cartilage in fifteen hips. Because of the frequency of the occurrence and poor prognosis, this complication should be more widely recognised.

TABLE III

THE FINDINGS IN FIFTEEN HIPS WITH ARTICULAR CARTILAGE NECROSIS AFTER SLIPPING OF THE UPPER FEMORAL EPIPHYSIS

Initial findings	Number of cases
Long history of symptoms	8
Short history of symptoms	6
Severe displacement	10
Slight displacement	5
Diminished joint space	5
Normal joint space	10

This complication presents in many respects like an inflammatory lesion of the hip; indeed, for a time two of the hips in this series were suspected of being tuberculous until disproved by biopsy. However, in all patients in whom the temperature, white cell count, and blood sedimentation rates were recorded they were normal. Four of the fourteen patients had arthrodesis performed within one year of first being seen; in three of these an intra-articular operation revealed necrosed articular cartilage but with healthy bleeding cancellous bone beneath it. The finding of this normal bone beneath the necrotic cartilage tends to confirm that the articular cartilage necrosis was not caused by a diminished blood supply. Waldenström expressed this view in 1930 and he pointed out that the articular cartilage of the acetabulum is also necrosed although its blood supply is not affected by the slipping of the femoral epiphysis. Also, as Moore (1945*b*) observed in his patient with bilateral necrosis of the articular cartilage, the epiphysis takes part in the disuse atrophy and appears rarefied, indicating that the blood supply is intact. Moore considered the cause to be severe non-specific inflammatory reaction, whereas Wilson (1949) thought it due to a partial loss of blood supply. Waldenström (1930) believed the explanation lay in the damage done to the synovial and capsular attachments superiorly by manipulation with resultant loss of synovial secretion.

FIG. 13

Case 20—A girl of fifteen who presented with a nine months' history of limping had a fibrous ankylosis, because of necrosis of articular cartilage which occurred naturally and before treatment.

The early findings in the patients in this series are shown in Table III. It will be seen that in five hips the joint space was already diminished at the first examination, and one of these

was already ankylosed (Case 20, Fig. 13). Three cases in the literature also showed evidence of loss of joint space on admission, and this suggests that sometimes the prognosis is poor regardless of the type of treatment.

Figure 14 shows the combination of treatments used. When traction—which varied from skin traction to a powerful skeletal pull—was prolonged for more than seven weeks it was also considered to be immobilisation in the classification of treatment. Thus immobilisation varied from rigid fixation in plaster or on a frame to skin traction in a Thomas's splint, which—although it allows a limited amount of movement—cannot be regarded as mobilisation.

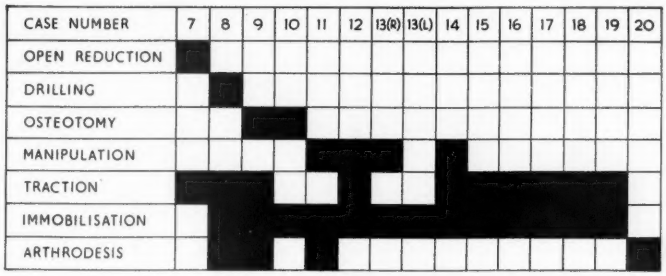


FIG. 14

The combination of treatment used in the fifteen hips with articular cartilage necrosis.

CONSIDERATION OF ADVERSE FACTORS IN TREATMENT

Immobilisation—It is of special interest that necrosis of the articular cartilage developed in two so-called " healthy " hips while the opposite hips were being treated for a slip. In both cases the " healthy " hip was immobilised only during the treatment of the opposite hip. In Case 19 the right hip first developed a slip and was treated by manipulation and immobilisation on a frame for nine months. It appeared to do well and the child had walked on it for a year when the left femoral epiphysis slipped. He was placed on an abduction frame for three months and during this time developed loss of joint space and permanent loss of movement in the right hip. The second, Case 13 (Figs. 15 to 17), was a girl who sustained a severe slip of the right femoral epiphysis which was treated by manipulation and immobilisation in a double spica for six months. Both hips showed marked loss of joint space and porosity of the head, and movements remained greatly restricted despite mobilisation for twelve months.

Moore (1945*b*) described a similar patient who had bilateral epiphysial displacement. Both hips were treated by traction and plaster, and both developed fibrous ankylosis. Jerre (1950) also reported an analogous case in which the unaffected hip became stiff, with loss of joint space. Hall (1957) reported a patient with bilateral slipping who showed loss of joint space in both hips after being on an abduction frame for ten weeks.

Twelve of the fifteen hips in this series were treated by immobilisation for over eight weeks, and in Jerre's (1950) series six of the nine patients showing articular cartilage necrosis had been treated by immobilisation and one by traction for eight weeks. Also the three original patients described by Waldenström (1930) were treated by immobilisation in plaster after manipulation. If the articular cartilage derives its nutrition from synovial fluid it may be assumed that rigid immobilisation, by producing atrophy of the synovial membrane and diminished synovial secretion, causes impaired nourishment of the articular cartilage. Müller (1929) suggested that nutrition of the articular cartilage was affected by the alternate physical processes of suction and pressure, which drew and expelled fluid from the adjacent network.

The recent biochemical studies on articular cartilage by Eichelberger, Roma and Moulder (1959) are important. Experimental cartilage atrophy, produced by denervation and disuse,

FIG. 15

FIG. 16

FIG. 17

Case 13—A girl of eleven with bilateral necrosis of articular cartilage. The radiograph on admission (Fig. 15) shows a severe slip on the right, but only slight on the left. The right hip was manipulated, and a double plaster spica applied, and removed after nine months (Fig. 16). After a further ten months of mobilisation the joint space on both sides is very poor (Fig. 17).

was studied. Simple immobilisation of a joint produced atrophy of the articular cartilage with changes in its physical state, particularly in its extracellular component. Disuse atrophy of articular cartilage was reversible, and the authors believed that this was explained by the viability of the chondrocytes. This in turn was controlled by several factors, one of which was chondroitin-sulphate production. But this is not yet fully understood. If it is linked with synovial fluid production, then there may be a point when the synovial tissue becomes so atrophied by prolonged immobilisation that the changes in the articular cartilage are no longer reversible.

Howorth (1949) reported the frequent finding of non-specific inflammatory changes in the synovial membrane after slipping of the epiphysis. It is possible that these inflammatory changes contribute to the impairment of the nutrition of the articular cartilage.

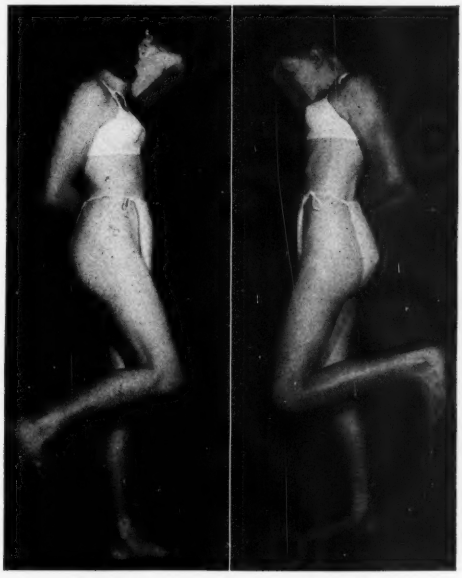


FIG. 18 FIG. 19

Case 13—The movements after mobilisation for ten months.

Traction—Traction in some form was used in ten of the fifteen hips which developed necrosis of the articular cartilage, and the head was distracted from the acetabulum in three hips which must have damaged the vessels in the ligamentum teres and in the synovial and capsular attachments. One hip was distracted with

FIG. 20 FIG. 21

Case 9—A boy of sixteen with recent severe displacement of the epiphysis. Figure 20—Initial film before treatment by traction and later by subtrochanteric osteotomy. Figure 21—Appearance nine months later. There was fibrous ankylosis.

nly fifteen pounds pull. Hall (1957) showed that traction alone produced very little correction n a slip, and the importance of its harmful influence has not been sufficiently recognised.

arly osteotomy—This was used in one patient (Case 9, Figs. 20 and 21) in this series. This

boy of sixteen had a severe slip partially corrected by traction and the remaining varus deformity corrected by a subtrochanteric osteotomy six weeks later. The hip was immobilised in a plaster spica for fifteen weeks. Nine months after this there was considerable loss of joint space with a fibrous ankylosis. With early osteotomy there is not only the danger that the operation may damage further an already precarious blood supply but also that it necessitates prolonged immobilisation. Osteotomy should, therefore, be deferred until a good range of movement has been obtained.

DISCUSSION

Early necrosis of the articular cartilage after slipping of the upper femoral epiphysis gives a very poor prognosis. Four of the hips in this series were arthrodesed within twelve months of first presenting. Probably the most important cause of this poor outlook is the loss of the smooth articular cartilage, but there may also be fibrosis in the capsule. The latter was demonstrated by Howorth (1949) in what he called the "residual stage" after slipping of the epiphysis. While it is accepted that good results can be obtained by a variety of methods of treatment, it must also be remembered that in any large series there will be some failures, and that there is no way in which these poor results can be predicted. It is difficult to understand why two hips with similar slips and similar treatment should give different results; this and many other aspects of the problem remain unsolved. Perhaps the explanation lies in the individual response to injury at an age when so many body changes are occurring as growing tissues alter to the mature adult state. Anatomical variations may also account for some of the differences.

SUMMARY AND CONCLUSIONS

1. Avascular necrosis of the bony epiphysis or necrosis of the articular cartilage of the hip joint—without bony necrosis—can occur after a slipped upper femoral epiphysis.
2. In avascular necrosis of the bony epiphysis the prognosis depends upon the degree of revascularisation that occurs and upon survival of the articular cartilage. The articular cartilage can survive and a good functioning hip result especially if aided by mobilisation without weight bearing.
3. The prognosis after necrosis of the articular cartilage is poor. This complication occurs more often when conservative treatment is used.
4. A certain number of hips will show poor results no matter what treatment is used.
5. Nutrition of the articular cartilage is probably by the synovial fluid.
6. Strong traction may damage the soft-tissue structure of the hip joint.
7. It is not advisable to perform an osteotomy soon after a slip of the epiphysis. It is better to wait until good function is assured in the joint.

I wish to thank the surgeons at the Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry, and the Prince of Wales Orthopaedic Hospital, Cardiff, for permission to study their patients.

In particular I am grateful to Mr Dillwyn Evans, Mr E. Mervyn Evans and Mr Robert Roaf for their helpful criticism and encouragement. I would also like to thank Mr Beverley Southern and Mr G. Haddock for the photographs.

REFERENCES

AXHAUSEN, G. (1922): Die Nekrose des proximalen Bruchstücks bein Schenkelhalsbruch und ihre Bedeutung für das Hüftgelenk. *Archiv für Klinische Chirurgie*, **120**, 325.

BAUER, W., ROPES, M. W., and WAINE, H. (1940): The Physiology of Articular Structures. *Physiological Reviews*, **20**, 272.

EICHELBERGER, L., ROMA, M., and MOULDER, P. W. (1959): Biochemical Studies of Articular Cartilage. III. Values Following the Immobilization of an Extremity. *Journal of Bone and Joint Surgery*, **41–A**, 1. 7.

HALL, J. E. (1957): The Results of Treatment of Slipped Femoral Epiphysis. *Journal of Bone and Joint Surgery*, **39–B**, 659.

HARRIS, W. R., and HOBSON, K. W. (1956): Histological Changes in Experimentally Displaced Upper Femoral Epiphyses in Rabbits. *Journal of Bone and Joint Surgery*, **38–B,** 914.

HOWORTH, M. B. (1949): Slipping of the Upper Femoral Epiphysis. *Journal of Bone and Joint Surgery*, **31–A,** 734.

JERRE, T. (1950): A study in slipped upper femoral epiphysis. *Acta Orthopaedica Scandinavica.* Supplementum No. 6.

JONES, R. Watson, and ROBERTS, R. E. (1934): The Radiological Significance of the Density of Bone Shadows. *British Journal of Surgery*, **21,** 467.

LANDELLS, J. W. (1957): The Reactions of Injured Human Articular Cartilage. *Journal of Bone and Joint Surgery*, **39–B,** 548.

MOORE, R. D. (1945*a*): Aseptic necrosis of the capital femoral epiphysis following adolescent epiphyseolysis. *Surgery, Gynecology and Obstetrics*, **80,** 199.

MOORE, R. D. (1945*b*): Conservative management of adolescent slipping of the capital femoral epiphysis. *Surgery, Gynecology and Obstetrics*, **80,** 324.

MÜLLER, W. (1929): *Biologie der Gelenke.* Leipzig: Barth.

PONSETI, I., and BARTA, C. K. (1948): Evaluation of Treatment of the Slipping of the Capital Femoral Epiphysis. *Surgery, Gynecology and Obstetrics*, **86,** 87.

TRUETA, J. (1957): The Normal Vascular Anatomy of the Human Femoral Head During Growth. *Journal of Bone and Joint Surgery*, **39–B,** 358.

WALDENSTRÖM, H. (1930): On Necrosis of the Joint Cartilage by Epiphyseolysis Capitis Femoris. *Acta Chirurgica Scandinavica*, **67,** 936.

WALDENSTRÖM, H. (1934): Necrosis of the Femoral Epiphysis. *Acta Chirurgica Scandinavica*, **75,** 185.

WATSON-JONES, R. (1943): *Fractures and Joint Injuries.* Third edition. Vol. 1, p. 80. Edinburgh: E. & S. Livingstone Ltd.

WILSON, P. D. (1949): Treatment of Slipped Epiphysis. (Discussion.) *Journal of Bone and Joint Surgery*, **31–A,** 21.

ZEMANSKY, A. P. Jun., and LIPPMANN, R. K. (1929): The Importance of the Vessels in the Round Ligament to the Head of the Femur During the Period of Growth, and their Possible Relationship to Perthes' Disease. *Surgery, Gynecology and Obstetrics*, **48,** 461.

BONE GROWTH IN DIAPHYSIAL ACLASIS

L. SOLOMON, LONDON, ENGLAND

From the Royal National Orthopaedic Hospital, London, and the Hospital for Sick Children, Great Ormond Street, London

An example of cartilaginous exostoses—believed by some to be the first described in detail in the literature—was reported in the Guy's Hospital Reports of the *Lancet* of July 2., 1825. However, it is certain that the condition was known at least half a century before that. In the series of Lectures on the Principles of Surgery which John Hunter delivered in 1786 and 1787 he described the condition of exostosis and distinguished between those that affect one bone and those that affect " almost every bone in the body." The bone ends might be enlarged " as in rickets," which observation led him to suppose that the two conditions were closely related.

There are more numerous references to the condition in the literature of the late nineteenth century, among which the classic papers of Virchow (1876) and Bessel-Hagen (1891) are remarkably detailed and accurate. In all these descriptions, as in those of recent times, interest centred mainly on the multiple cartilage-capped exostoses which are so striking and curious a feature of the disease.

However, an equally important and perhaps a more fundamental aspect of this malady is the widespread effect on the growth of the appendicular skeleton. It was this, above all, that impressed Keith (1920) when he first encountered the condition and led him to adopt the name " diaphysial aclasis," suggested to him by Morley Roberts and still commonly used in this country. He urged that the condition of multiple exostoses be removed altogether from the category of " tumours " and placed among the " disorders of growth." In spite of this, bone growth in diaphysial aclasis has received scant attention. The present investigation forms part of a detailed study of hereditary multiple exostoses carried out at the Royal National Orthopaedic Hospital and the Hospital for Sick Children, Great Ormond Street. The findings are presented in the hope that they will stimulate further study on what is probably the basic defect in this puzzling disease.

MATERIAL AND METHODS

Eighty-nine cases of diaphysial aclasis have been studied, and of these seventy-six were fully examined by the author. The examination included a full radiographic survey of the skeleton and detailed measurements of height, weight, span, symphysial height and measurement of individual long bones. The skeletal age of every patient under eighteen was estimated against the standards of Greulich and Pyle (1959). When previous radiographs of the hand and wrist were available the skeletal age was estimated from these also, and correlated with the chronological age at that time. There are other methods of estimating skeletal maturity—all based on the characteristic appearances of the epiphysial ossification centres at different ages—but the method of Greulich and Pyle offers considerable advantages in a study of a generalised bone disease such as diaphysial aclasis. The reference standards consist of a large series of radiographs of the hand and wrist taken at different ages, showing not only the evolution of ossification centres in the carpal bones but also the slowly changing pattern of the individual bones of the hand as well as the distal ends of the radius and ulna. In this way one can compare skeletal maturation in the tubular bones of the hand, the radius and the ulna (all sites frequently affected by diaphysial aclasis), with the carpal bones, which are very rarely affected by exostoses.

In plotting the height distribution of these patients standard graphs have been constructed using Tanner's (1958) data for normals up to the age of eighteen, and Kemsley's (1950) figures for normal adults. The problems involved in obtaining normal standards have been discussed at length by Tanner (1958) and will not be repeated here. Although Tanner's and Kemsley's figures for normal adolescents do not coincide, it was decided to show these measurements as a single broken graph rather than as two separate graphs.

Much more difficult to obtain were normal figures for span, symphysial height (" lower measurement ") and symphysis-to-vertex (" upper measurement "). Yet it is important to present these figures—and particularly the comparative upper and lower measurements—because they are an indication of the extent to which limb length is affected by comparison with the trunk. The most reliable standards (though unfortunately presented only as mean values) were those of Engelbach (1932), and they are the ones used in this paper.

Finally, in order to minimise the possible errors due to using standards which differ from each other as to population measured and the time of these measurements, a comparable series of " healthy " out-patients attending the Royal National Orthopaedic Hospital were measured as well and checked against the standards used.

The findings are discussed under three main headings: 1) *Abnormal bone growth:* retardation of bone growth; and adaptations of bone growth due to pressure by adjacent exostoses. 2) *Migrating exostoses.* 3) *Disappearing exostoses.*

ABNORMAL BONE GROWTH

The patient shown in Figure 1 presented simultaneously all the deformities encountered in diaphysial aclasis. Though by no means a dwarf he had the characteristically short stature, being 157 centimetres in height. The left forearm, in addition to being short, showed bowing of the radius, ulnar deviation of the wrist due to a short ulna, and dislocation of the radio-humeral joint. The left hand was broad and stubby, the fingers shorter than those of the right hand. There was genu valgum on the right side and a valgus deformity of the right ankle. There was a mild though definite thoracic scoliosis with vertebral rotation. On radiographic examination the pelvis was distorted and there was bilateral coxa valga. All these deformities, either singly or in varying combinations, have been described before, but there were two further deformities that do not appear in the literature on the subject. Firstly, the left clavicle was clearly diminished in length compared with the right side and on measurement was almost two centimetres shorter than the right. This gave rise to the asymmetrical appearance of the pectoral girdle. Secondly, the thoracic cage under the right scapula presented a gentle indentation where it had moulded itself to a large exostosis on the deep aspect of the scapula (Fig. 2).

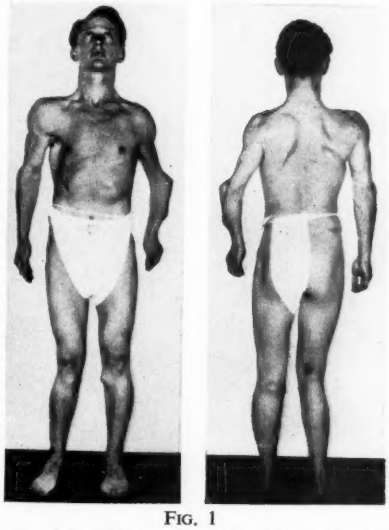


FIG. 1
The deformities associated with diaphysial aclasis.

Incidence and distribution of deformities—Bony deformities of one sort or another (as distinct from exostoses) were encountered in fifty-six of the seventy-six patients examined in detail, and were distributed equally as to sex (Table I). These fifty-six patients were analysed in greater detail and the occurrence of individual deformities is shown in Table II. No attempt has been made to present figures for conditions like shortening of the clavicles and coxa valga which are in most cases difficult to assess.

Of all these characteristic deformities only the valgus ankles have any clear tendency to be symmetrical. The others are scattered haphazardly in one limb or another, though there is some relationship to the number and size of the exostoses affecting the particular region. When a bone is free from any exostosis an associated deformity is rare.

RETARDATION OF BONE GROWTH

All the deformities of the tubular bones can be explained in terms of the same common factor: diminished length of the bones affected by the disease. A closer look at the individual deformities will illustrate this point.

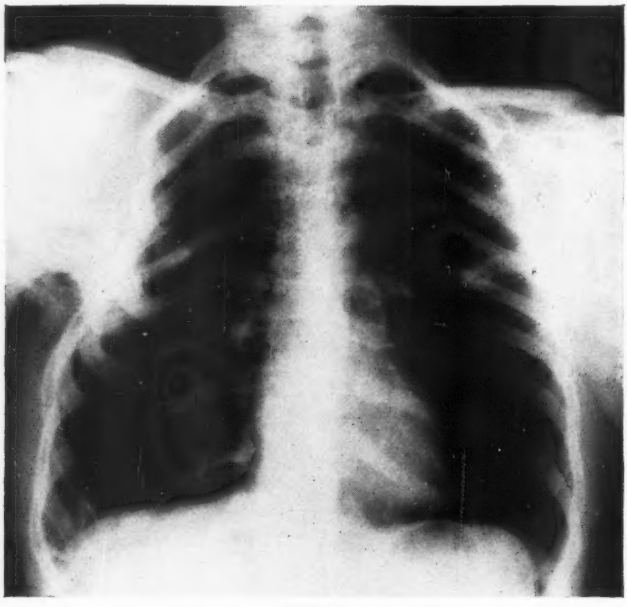


FIG. 2
Radiograph showing deformity of thoracic cage on right side.

Deformities of the forearm and wrist—One or other of the characteristic forearm deformities occurred in almost half of the patients examined, though the more marked manifestations such as radio-humeral dislocation were comparatively uncommon. Figure 3 shows the radiographs of one such patient. The radius is bowed throughout its length, not merely at the distal end where exostoses are common. Moreover, the left radius is bowed though this bone has no exostosis at all.

The gravest effect here is obviously the gross shortening of the ulna. Even when both bones are equally affected by exostoses the ulna is almost invariably more severely shortened than the radius. This growth deficiency is confined to the distal end of the bone, which is typically carrot-shaped; the radio-ulnar joint is often disrupted and the wrist deviated to the ulnar side.

There are at least two reasons for this disproportionate shortening of the distal end of the ulna. Exostoses are rare at the proximal end of either the radius or the ulna, whereas the distal ends are affected in 85 and 68 per cent of cases respectively. This is in keeping with the general observation that the bone ends which contribute most to the total diaphysi-l

TABLE I

INCIDENCE OF DEFORMITIES IN SEVENTY-SIX PATIENTS WITH DIAPHYSIAL ACLASIS

Sex	Multiple exostoses	Associated deformities	Percentage with deformities
Males	36	26	72
Females	40	30	75
Total	76	56	73·7

TABLE II

THE DEFORMITIES PRESENT IN SEVENTY-SIX PATIENTS WITH DIAPHYSIAL ACLASIS

Deformity	Number of patients				Percentage
	Both sides	Right side	Left side	Total	
All forearm deformities	15	8	14	37	50
Bowed radius	13	7	13	33	43
Conical ulna	5	6	8	19	25
Radio-humeral dislocation	1	2	3	6	8
Genu valgum	7	3	6	16	21
Valgus ankles	29	4	1	34	45
Deformities of the hands	6	3	4	13	17
Short stature	—	—	—	31	41
Scoliosis	—	—	—	3	4
Pelvic deformities	—	—	—	3	4
Thoracic deformities	—	—	—	2	3

TABLE III

THE RELATIONSHIP BETWEEN RATE OF GROWTH AND FREQUENCY OF EXOSTOSES (SEVENTY-SIX CASES)

Bone	Proportionate contribution to bone length (expressed as a percentage of total elongation)		Number of cases affected	Percentage of total
Femur	Proximal end	20	58	77
	Distal end	80	71	94
Tibia	Proximal end	60	72	95
	Distal end	40	62	82
Fibula	Proximal end	55	69	91
	Distal end	45	55	72
Humerus	Proximal end	80	65	85
	Distal end	20	3	4
Radius	Proximal end	25	10	13
	Distal end	75	64	85
Ulna	Proximal end	15	7	9
	Distal end	85	52	68

The figures for the proportionate contribution of the proximal and distal ends to bone length are after Lacroix (1951).

length are the more frequently affected in diaphysial aclasis (Table III). Although it is almost impossible to determine the exact proportionate contribution of proximal and distal ends to total bone length (indeed, it varies quite widely from person to person), it seems certain that the distal end of the ulna contributes more to total bone length than the distal end of the radius. Equal involvement of these two bones at more or less the same age can therefore be expected to cause a greater diminution in the ultimate length of the ulna than of the radius.

FIG. 3

Radiograph showing the characteristic forearm deformities. The ulna is short and the distal end tapered; the radius is bowed throughout its length and there is subluxation of the right radio-humeral joint.

The second possible reason for this undue shortening of the ulna is the difference in cross-sectional area of the epiphysial plates of the two bones, the ulnar plate being less than a quarter the area of the radial plate. " Equal " involvement by exostoses, therefore, means greater proportionate involvement of the ulnar growth plate. Whatever the ultimate mechanism may be, there is evidence that the retardation of growth is related to the extent to which the particular epiphysial plate is disturbed by the disease.

The result of this disproportionate ulnar shortening is that the radius, tethered firmly to the ulna and growing considerably more than the latter, is accommodated in one of two ways: either it bends or there is dislocation of the radio-humeral joint. When dislocation occurs the bowing is less than would otherwise be expected (Fig. 4).

Valgus deformity of the ankle and knee—Obliquity of the tibial articular surface with valgus tilting of the talus occurred in thirty-four of the seventy-six patients, and of these all but five were bilateral and symmetrical (Fig. 5). It is important to notice that the lower epiphysial plate of the tibia remains horizontal; it is not distorted except in the most severe cases, and even then the horizontal disposition is retained though the lateral part of the articular surface is forced right into the metaphysis.

The valgus deformity is almost entirely a deformity of the tibial epiphysis, which is wedge-shaped in all these cases. The underlying reason for this is much the same as with the forearm deformity, namely, a disproportionate degree of shortening of the fibula, and the possible causes of this shortening are the same as those already discussed. The lateral malleolus comes to lie at the same level as—and sometimes higher than—the medial malleolus, though still retaining its strong attachment to the tarsus through the ligaments of the ankle joint. The talus is thus tilted into valgus and drawn up hard against the lateral part of the tibial articular surface, which increasingly bears the stress due to the disproportionate tibial growth. Growth of the tibial epiphysis, which is derived almost entirely from the articular cartilage, is thus interfered with mainly in its lateral half, with consequent wedging of the epiphysis. If the deformity were due to unequal growth at the tibial epiphysial plate, this plate would itself be tilted and distorted obliquely, and this has been shown not to occur.

The same argument applied to the upper end of the fibula and the knee accounts for the valgus deformity of the knee in at least a certain proportion of patients. In others the tibia, attempting to accommodate itself to the shortened fibula, may actually bend in much the same

FIG. 4

Dislocation of the radio-humeral joint. The projection at the elbow is not an exostosis; it is due to the proximal end of the radius which has been dislocated since childhood.

FIG. 5

Radiographs showing valgus deformities of the ankles. Note that in spite of the marked tilting of the tibial articular surface the line of the epiphysial plate is horizontal.

way as the radius becomes bowed (Fig. 6). Thus, whatever the particular form of the resulting deformity, the inescapable conclusion is that it results from diminished bone length, the " short bone " in this instance being the fibula.

Shortening of the clavicle—Like other bones affected by multiple exostoses, the clavicle may be shorter than usual (Fig. 1). It remains only to comment on the fact that this bone should be affected at all. Regarded by many as a membrane bone, it was at one time thought not to be involved in diaphysial aclasis—a disease of the bones preformed in cartilage. Routine radiographs, however, show that the clavicle is involved in 24 per cent of patients with this disease, usually at its acromial end, but sometimes also at its sternal end.

FIG. 6

Radiograph showing genu valgum from bowing of the tibiae. There is also a tibio-fibular synostosis on the left side.

The development of the clavicle has been studied in detail (Fawcett 1913, Hanson 1920, Todd and D'Errico 1928, Gray's Anatomy 1958), and it is not surprising to learn that although the bone is developed directly from the primitive mesenchyme, precartilaginous (and later cartilaginous) elements develop at the sternal and acromial ends shortly before these regions become ossified. There is therefore no reason why the clavicle should not be involved like any other bone that develops through a cartilaginous phase.

Short stature—Some of the earliest descriptions of multiple exostoses comment on the fact that these patients tend to be shorter than normal. This is a general impression which has not been substantiated by detailed anthropometric studies in large series.

The present group of patients were measured for total height, span, symphysial height and length of individual long bones.

The height distribution of these patients is shown in Figures 7 and 8. The standards used were derived from two sources. Up to the age of eighteen Tanner's standards are the most reliable, and a corresponding series of " healthy " children and adolescents attending the hospital as out-patients were found to be evenly distributed along these curves. For the adults Kemsley's standards have been chosen as the most reliable and representative of the studies on British people. It is likely that the present population is slightly taller than is reflected in these standards, obtained before 1943. A series of " healthy " out-patients corresponding in age and sex to the patients with diaphysial aclasis was also measured, and compared with Kemsley's standards they all tend to be slightly taller. The fiftieth percentile in this group falls almost exactly on the seventy-fifth percentile of Kemsley's series. The values shown in Figures 7 and 8 are therefore, if anything, even lower than they seem compared to the normal.

There are several points worthy of comment in these figures. 1) The diminution in height, though undoubtedly evident, is not on the whole great. Only seven patients fell below the third percentile and can be regarded as highly abnormal. 2) The shortness of stature becomes more evident as the end of the growth period is reached. Thus, before puberty there is hardly any deviation from the normal, but from then onwards growth is clearly retarded by comparison

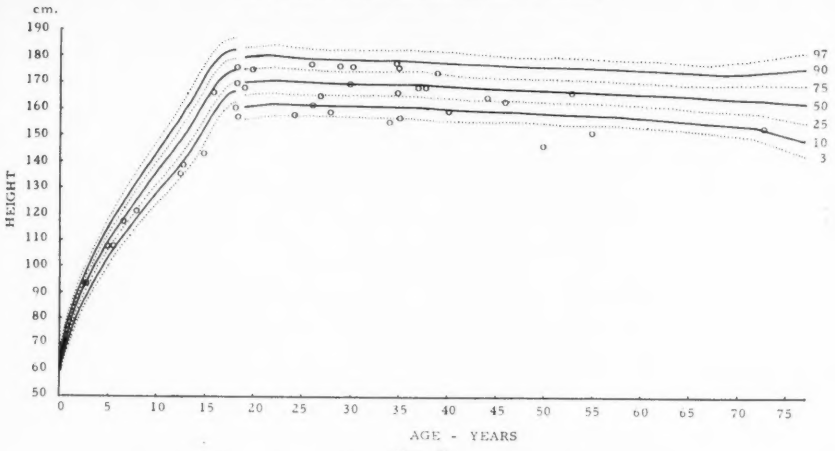


FIG. 7

Analysis of the height distribution in patients with diaphysial aclasis (males). The standard curves are derived from Tanner (1958) and Kemsley (1950), the figures on the right showing the third, tenth, twenty-fifth, fiftieth, seventy-fifth, ninetieth and ninety-seventh percentiles.

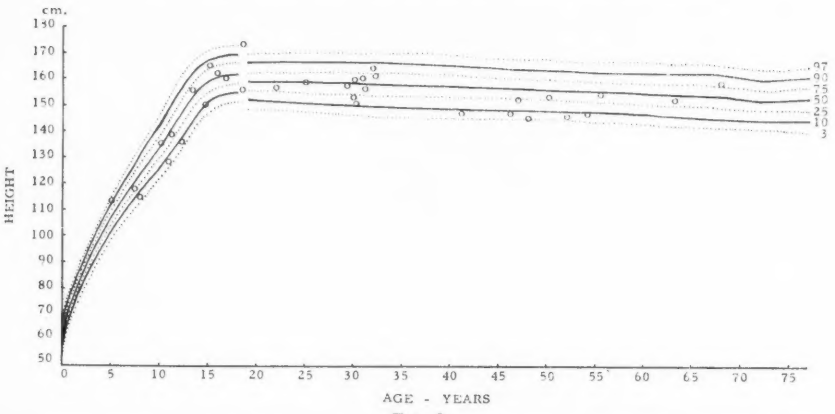


FIG. 8

The height distribution in patients with diaphysial aclasis (females). The standard curves are derived from Tanner (1958) and Kemsley (1950), the figures on the right showing the third, tenth, twenty-fifth, fiftieth, seventy-fifth, ninetieth and ninety-seventh percentiles.

with the normal. 3) Males, on the whole, are more severely retarded than females. This could be explained by the fact that girls reach the end of the growth period earlier than boys and the effects of diaphysial aclasis on growth continue for a considerably longer period in the latter.

As might be expected, this diminution in stature is almost entirely due to shortness of the lower limbs, the trunk being more or less normal. Figures 9 and 10 show the upper and lower measurements in these cases compared with Engelbach's standards (1932). Here again the possible errors were minimised by measuring "healthy" out-patients and the mean values were found to correspond closely to Engelbach's figures, obtained before 1932 mainly on native Americans. The symphysial height is a reliable index of total lower-limb length.

The span, from finger tip to finger tip, measures not only the combined length of the long bones but also that of the hands and the pectoral girdle. These measurements are shown

in Figures 11 and 12, the standards again being those of Engelbach. Retardation of growth of the tubular bones is reflected to a much greater extent here than in either the symphysial height or the total height.

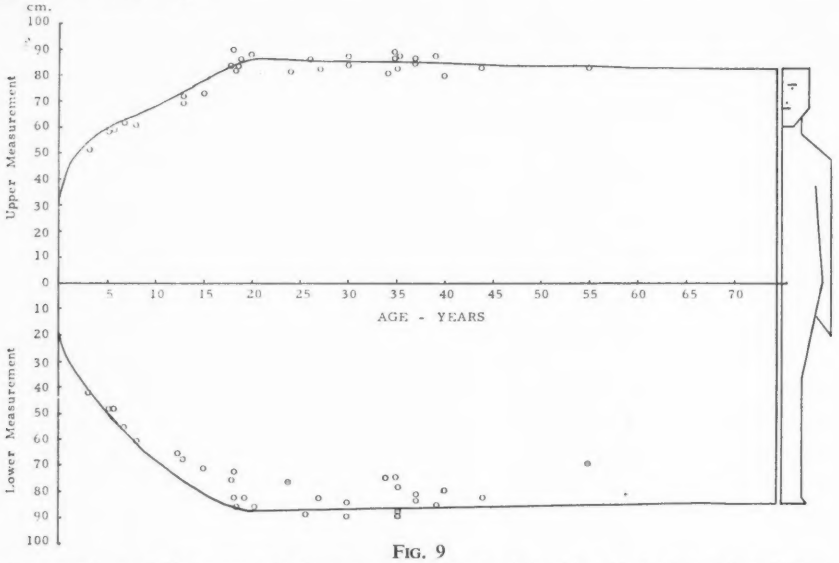


FIG. 9

A comparison of the upper and lower measurements (males). The standards are derived from Engelbach (1932).

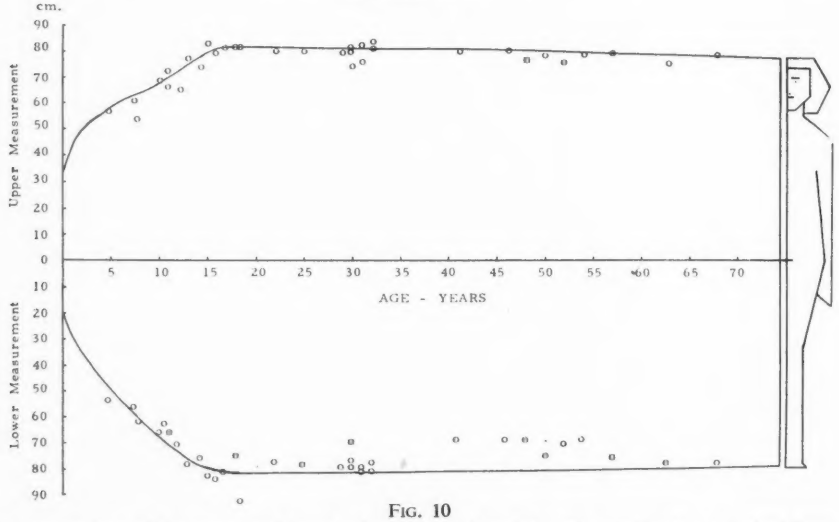


FIG. 10

A comparison of the upper and lower measurements (females). The standards are derived from Engelbach (1932).

There are no reliable clinical standards for comparing the length of the individual bones, but an interesting impression was gained from studying these measurements. The degree of shortening in the long bones tends to be progressively more and more severe in the following order: the femur least of all, then tibia, humerus, radius, fibula and the ulna most severely

of all. This is exactly in keeping with the suggestion made previously that the degree of shortening is related to the cross-sectional area of the epiphysial plates of the bones affected, the narrowest bones being shortened the most and the widest the least.

Pathogenesis—The future pattern of growth in these cases is quite unpredictable when exostoses first appear. Figures 13 and 14 show the progress of the disease in one child over a period of

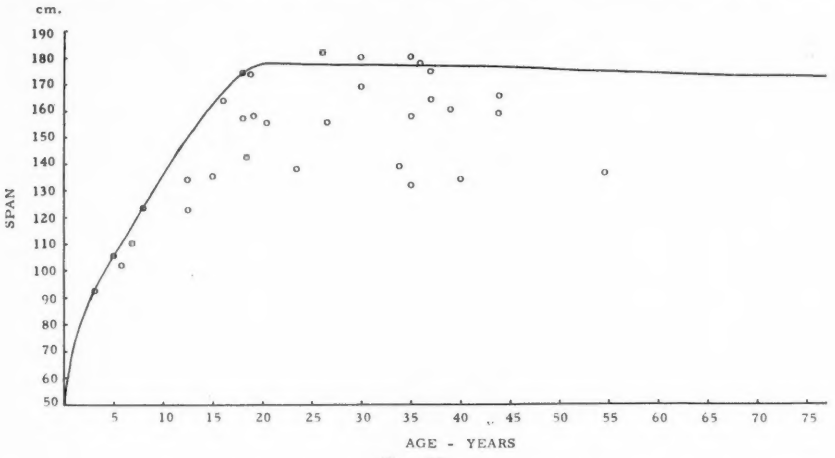


FIG. 11

The measurement of span in diaphysial aclasis (males). The standards are derived from Engelbach (1932).

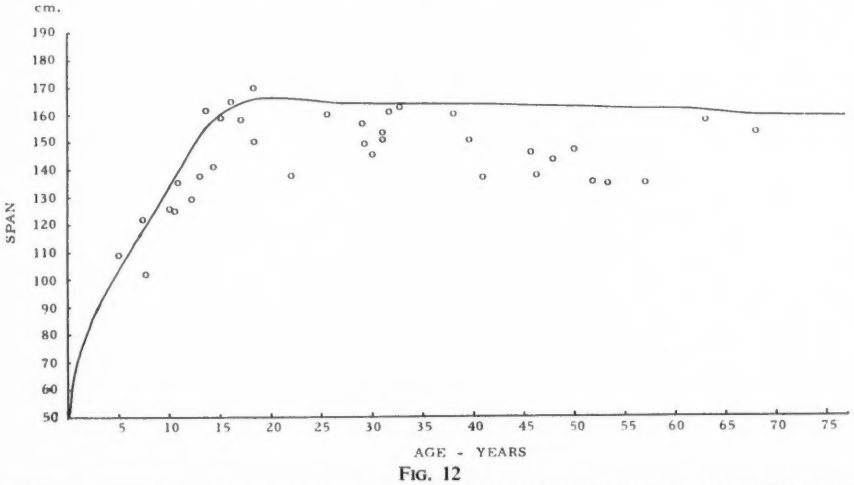


FIG. 12

The measurement of span in diaphysial aclasis (females). The standards are derived from Engelbach (1932).

ten years from the age of three to thirteen. The earliest exostosis at the proximal end of the humerus appeared during the same year as the exostosis at the distal end of the radius. During the next ten years of growth the proximal end of the humerus has continued to grow in its new, grossly abnormal mould with considerable shortening, while over the same period all further growth of the distal end of the radius has been absolutely normal and at the age of thirteen the original exostosis is hardly recognisable half way along the shaft of the bone.

The cause of this retardation in the growth of the long bones in diaphysial aclasis cannot be separated from the cause of the disease as a whole. There is no evidence that it is due to precocious skeletal maturation or early closure of the epiphysial plates, though this belief is still quite widely held (Illingworth and Dick 1956). Figure 15 shows that skeletal maturation in these patients is normal and the epiphyses close at the normal times, whether the bones are affected by multiple exostoses or not.

FIG. 13
Radiographs of the left humerus showing the progress of diaphysial aclasis over ten years.

The alternative theory of "squandered growth potential," though couched in less scientific terms, is much more attractive (Jaffe 1943). This theory not only recognises the connection between the occurrence of exostoses and the retardation of growth in the affected bone, but actually regards the one thing as being dependent on the other. The appearance of these abnormal bones suggests that the normal growth potential, which should have been directed to increasing the length of the bone, has been squandered on the formation of abnormally broad metaphyses and multiple exostoses. Certain it is that a bone which is markedly broadened in the metaphysial region, possibly with numerous exostoses as well, is invariably shorter than an unaffected opposite number; but whether the degree of shortening is proportionate to the overall thickening of the metaphysial region is impossible to determine unless the entire skeletal system is available for morbid anatomical studies at various ages during the period of growth.

Apart from emphasising the relationship between the diminution in bone length and broadening of the metaphyses with the formation of multiple exostoses, I have made no attempt here to discuss the etiology of hereditary multiple exostoses, which will form part of a further paper.

ADAPTATIONS OF BONE GROWTH DUE TO PRESSURE BY ADJACENT EXOSTOSES

Certain abnormalities of bone growth are induced simply by prolonged pressure from adjacent exostoses (Fig. 2). By the process of osteoclastic resorption and bone modelling the growing bone accommodates itself to the obtrusive exostosis in the characteristic way. This occurs quite frequently in the radius and ulna, the tibia and fibula and in adjacent ribs, but may

FIG. 14
Radiographs showing the radius and ulna in the same patient as Figure 13.

also be seen in almost any situation where a large exostosis abuts on a neighbouring bone, or even on the very bone from which it arises.

Tibio-fibular synostosis—Whereas a growing bone will mould itself in the manner described, if two adjacent exostoses abut against each other the results are quite different. If either of the two exostoses continues to grow, the one will gradually become embedded in the cartilaginous cap of the other and this may eventually go on to synostosis between the two bones (Fig. 6). This is distinctly unusual in the radius and ulna, where supination and pronation

movements probably prevent such an outcome, but tibio-fibular synostosis occurred in nineteen (or 25 per cent) of the seventy-six patients in whom complete radiographic examinations were carried out, and of these all but three were affected bilaterally. It is perhaps worth commenting that, apart from the fact that most of these patients also had valgus deformities of the ankles, function was otherwise undisturbed.

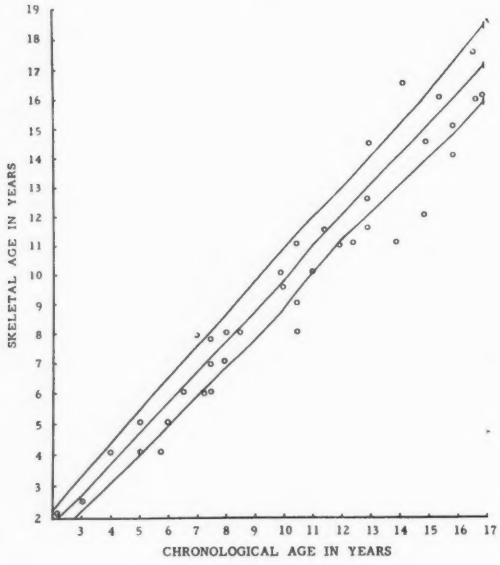


FIG. 15

Skeletal maturation in diaphysial aclasis. The standards are derived from the Harvard series quoted in Greulich and Pyle (1959) and are shown as the mean and one standard deviation above and below.

MIGRATION OF EXOSTOSES

The exostoses, once formed, may be thought to retain their relationship to each other, at least at their attachment to the diaphysial shaft. In studying the radiographs of children with multiple exostoses one is often struck by the fact that two easily identifiable exostoses—one at the proximal and one at the distal end of the bone—appear to move farther and farther apart over the years. Figure 16 shows two successive radiographs, the one taken at the age of fourteen and the other at the age of sixteen. In the earlier radiograph the exostoses E1 and E2 are 15·1 centimetres apart. In the later radiograph they are 16·8 centimetres apart. Only one other reference to this phenomenon has been discovered in a careful search of the literature (Lacroix 1950). Yet it is the rule rather than the exception for this to occur and it can be demonstrated in almost every case provided appropriate radiographs are taken at successive ages during the period of growth.

The fortuitous co-existence of " Harris's lines " in some of these cases has allowed a comparison with bone growth as a whole. Figure 16 shows that, whereas the distance between the exostoses increases, the distance between the lines G1 and G2 remains the same over the years. In other words, diaphysial length has remained unchanged, while the exostoses have moved farther and farther apart. Logically, there can be only one cause for this phenomenon—namely, progressive osteoclastic resorption on the diaphysial side and simultaneous osteoblastic new bone formation on the epiphysial side of each exostosis. Figures 17 and 18 show the histological features of one such case. This exostosis has

demonstrably altered its relationship to a nearby Harris's line by no less than 1·5 centimetres over the space of five years. When ultimately excised (on account of continuous pain) a small piece of the adjacent cortex was taken as well (Fig. 17). The base of the exostosis shows numerous osteoclasts in their lacunae along the margin facing towards the mid-shaft, and the bony trabeculae are being actively replaced by rows of osteoblasts massed along the opposite margins.

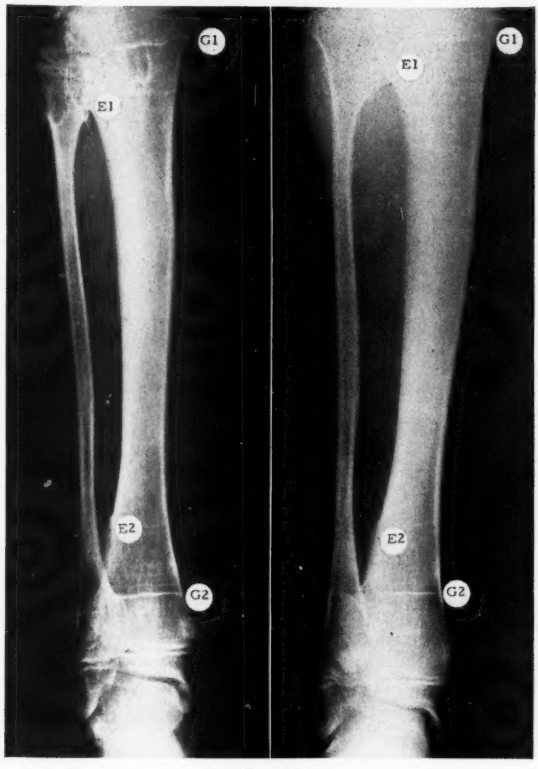


FIG. 16

Radiographs to show the migration of exostoses over a period of two years. The exostoses E1 and E2 have moved farther apart while the lines G1 and G2 have remained unchanged. (Left-hand radiograph aged fourteen years and right-hand radiograph at sixteen years.)

The stimulus for this osteoclastic resorption is possibly, as Lacroix suggests, the differential rate of growth (or " stretching ") of the periosteum over the diaphysial cortex as the overall length of the bone increases.

DISAPPEARING EXOSTOSES

An early reference to disappearing exostoses can be found in John Hunter's Lectures of 1786, published in 1835. Writing about the treatment of the condition he says: " It is hardly ever to be cured by medicines either external or internal, but as it is sometimes spontaneously removed we should endeavour to promote the absorption of it by rousing up this power."

About one-third of the adult patients say that one or more of the bony lumps which they had in childhood have gradually disappeared over the years. This raises an important question and might have a bearing on treatment. These statements were therefore investigated with particular care, and were substantiated in a surprising number of cases.

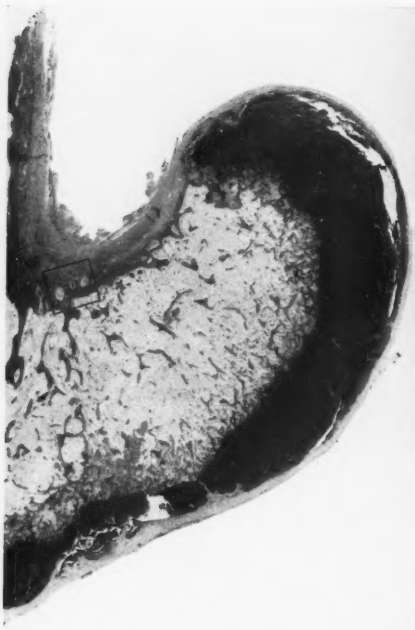


FIG. 17

FIG. 18

FIGS. 17 AND 18

Figure 17—Photomicrograph of a cartilage-capped exostosis showing the region where osteoclastic resorption and active new bone formation have been demonstrated. Figure 18—Photomicrograph of the area marked out in Figure 17. Osteoblasts are massed along the advancing edges of the bone trabeculae while the trailing edges are ragged with numerous Howship's lacunae. (The arrow shows the direction in which the exostosis is migrating.)

Figure 19 shows a typical example of disappearing exostoses. The exostosis at the base of the proximal phalanx of the left middle finger has disappeared completely by the age of ten: the smaller exostosis which shows at this age at the distal end of the proximal phalanx of the right middle finger has likewise disappeared by the age of thirteen and a half.

The explanation for this is probably quite simple. The overall diameter of the phalangeal shaft plus the exostosis in each case remains unchanged over the years. Clearly the exostosis must have ceased growing while the phalanx enlarged in the normal way, increasing its diameter by appositional growth until the overall dimensions obscured the once prominent exostotic projection.

SUMMARY AND CONCLUSION

1. The widespread deformities commonly associated with diaphysial aclasis have been studied in seventy-six patients. Apart from the adaptations of growth due to pressure by neighbouring exostoses, all the deformities of the tubular bones can be explained in terms of the same underlying factor—diminished length of the bones affected by the disease.
2. When the condition first manifests itself the future pattern of bone growth is completely unpredictable except in so far as it is known that the more actively growing ends of the long bones are the more severely affected in each case. It has also been shown in this series that, in general, the bones with the smallest cross-sectional area at the epiphysial plates (such as the ulna and the fibula) are the most severely shortened of all.
3. The cause of this disturbance of growth is still unknown, but there is an undoubted relationship between the presence of exostoses or thickening of the metaphysial region and shortening of the bone involved.
4. The phenomena of migrating exostoses and disappearing exostoses are also described and are shown to be examples of the normal process of bone modelling applied in special circumstances.

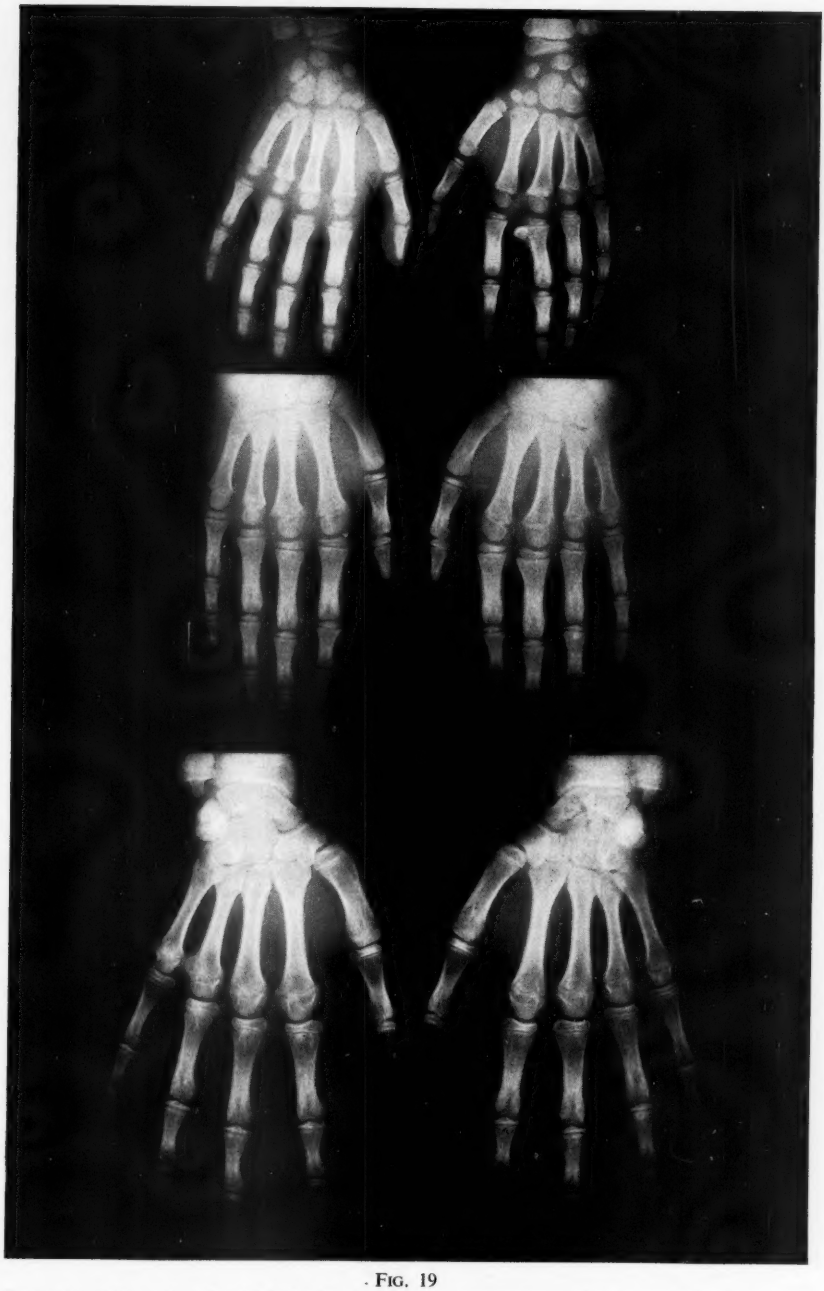


FIG. 19

Successive radiographs of a child's hands showing the disappearance of exostoses between the ages of five years and thirteen and a half years.

5. Although the importance of the cartilage-capped exostoses is not underestimated, it is hoped that this study will stimulate further work on what is probably the basic defect in this disease—namely, the disturbance of bone growth.

I should like to express my gratitude to Dr H. A. Sissons who first suggested this study and whose interest has been a constant encouragement. I am also grateful to the members of the staffs of the Royal National Orthopaedic Hospital and the Hospital for Sick Children, Great Ormond Street, for allowing me free access to their patients.

The photograph for Figure 1 was supplied by the Photographic Department of the Middlesex Hospital. All the other photographs were prepared by Mr R. J. Whitley, Mrs P. Thomas and Mr M. Duffett of the Photographic Department of the Royal National Orthopaedic Hospital.

I should like to thank Miss D. Beridge and Miss D. Hodgkin for their untiring help in arranging the follow-up clinics and preparing the manuscript and tables for this paper.

REFERENCES

BESSEL-HAGEN, F. (1891): Ueber Knochen- und Gelenkanomalieen. *Archiv für Klinische Chirurgie*, **41**, 420.

ENGELBACH, W. (1932): *Endocrine Medicine*. Vol. I. Springfield, Illinois: Charles C. Thomas.

FAWCETT, E. (1913): The Development and Ossification of the Human Clavicle. *Journal of Anatomy and Physiology*, **47**, 225.

GRAY'S ANATOMY (1958): Thirty-second edition. Edited by T. B. Johnston, D. V. Davies and F. Davies. London: Longmans, Green and Co.

GREULICH, W. W., and PYLE, S. I. (1959): *Radiographic Atlas of Skeletal Development of the Hand and Wrist*. Second edition. California: Stanford University Press.

HANSON, F. B. (1920): The History of the Earliest Stages in the Human Clavicle. *Anatomical Record*, **19**, 309.

HUNTER, J. (1835): *The Works of John Hunter, F.R.S.* With Notes by J. F. Palmer. Vol. I. London: Longman, Rees, Orme, Brown, Green & Longman.

ILLINGWORTH, C. F. W., and DICK, B. M. (1956): *A Textbook of Surgical Pathology*. Seventh edition. London: J. & A. Churchill Ltd.

JAFFE, H. L. (1943): Hereditary Multiple Exostosis. *Archives of Pathology*, **36**, 335.

KEITH, A. (1920): Studies on the Anatomical Changes Which Accompany Certain Growth-disorders of the Human Body. *Journal of Anatomy*, **54**, 101.

KEMSLEY, W. F. F. (1950): Weight and Height of a Population in 1943. *Annals of Eugenics*, **15**, 161.

LACROIX, P. (1950): Contribution a l'étude de la maladie exostosante. *Revue d'Orthopédie*, **36**, 20.

LACROIX, P. (1951): *The Organization of Bones*. (English Translation.) London: J. & A. Churchill, Ltd.

TANNER. J. M. (1958): The Evaluation of Physical Growth and Development. In *Modern Trends in Paediatrics*. Second Series. Edited by A. Holzel and J. P. M. Tizard. London: Butterworth and Co. (Publishers) Ltd.

TODD, T. W., and D'ERRICO, J., Jun. (1928): The Clavicular Epiphyses. *American Journal of Anatomy*, **41**, 25.

VIRCHOW, R. (1876): Ueber die Entstehung des Enchondroms und seine Beziehungen zur Enchondrosis und Exostosis cartilaginea. *Monatsberichte der Königlichen Preussischen Akademie der Wissenschaften*, p. 760.

TIBIALIS POSTERIOR TRANSFER IN CONGENITAL CLUB FOOT

MARTIN SINGER, CAPE TOWN, SOUTH AFRICA

In a review of the results of tibialis anterior transfer in congenital club foot it was found that fifty-two relapses occurred in a series of seventy-six tendon transfers (Singer and Fripp 1958). In addition certain undesirable sequelae resulted from the operation: 1) a dorsiflexed (cocked-up) hallux and dropped first metatarsal head in fifty-seven instances; and 2) disturbed muscle balance, the transposed muscle producing excessive pronation of the forefoot in eleven patients and excessive valgus of the hindfoot in three. The operation was based on the premise that in the presence of relative peroneal insufficiency the tibialis anterior muscle was the most powerful deforming factor causing relapse.

In all of the fifty-two relapses the principal component, and usually the only one, was the equinus deformity. The results suggested that the basic principle of the operation was incorrect, because the tibialis anterior is an important dorsiflexor of the foot as well as an invertor. In transferring it one might encourage the recurrence of equinus deformity because transfer always weakens a muscle. This tendency is aggravated by the power of the tibialis posterior muscle. The influence of this muscle is seen during the operation of soft-tissue correction (Brockman 1930), when invariably it must be lengthened to allow correction of the deformity. The power of the tibialis posterior was also demonstrated by Gunn and Molesworth (1957), who found it an effective dorsiflexor when transferred to the dorsum of the foot for lateral popliteal nerve paralysis in leprosy.

It was apparent that tibialis anterior transfer had an extremely limited place in the treatment of the difficult or relapsed club foot. An operative trial of transferring the tibialis posterior muscle through the interosseous membrane to the dorso-lateral aspect of the foot was undertaken. In this way a major deforming force would be removed and the tibialis posterior acting from its new insertion, although weakened, would supplement the relatively weak peroneal muscles and at the same time the dorsiflexor power of the tibialis anterior would be unimpaired. The retention of the tibialis anterior in its normal position might also prevent the development of the cocked-up hallux and a depressed first metatarsal head. This concept was supported in an editorial by Barr (1958).

CLINICAL MATERIAL

The patients in this series constitute the " difficult " group of club feet. They all had some residual deformity when treatment started and this was due to relapse of a previously corrected deformity or to incomplete initial correction or to lack of previous treatment. Eleven of the seventeen children were non-European. A total of twenty-eight feet in seventeen patients were corrected before operation by the Kite method (Fripp and Singer 1953). The ages of the patients varied from two and a half to eight and a half years at the time of the operation.

TECHNIQUE OF OPERATION

The tibialis posterior tendon is exposed through a curved incision behind the medial malleolus. The incision starts an inch above the tip of the malleolus and extends downwards as far as the tuberosity of the navicular bone. In every instance the tibialis posterior tendon was thicker than normal in this region, and in some patients the tendon had already split into two under cover of the malleolus. After detachment of the tendon as far distally as possible a strong stay suture is inserted into the cut end.

A second incision, two and a half to three inches long, is made at the junction of the middle and distal thirds of the tibia. The incision skirts the crest of the tibia on its lateral aspect. Through this incision, and extraperiosteally, the interosseous membrane is exposed and an

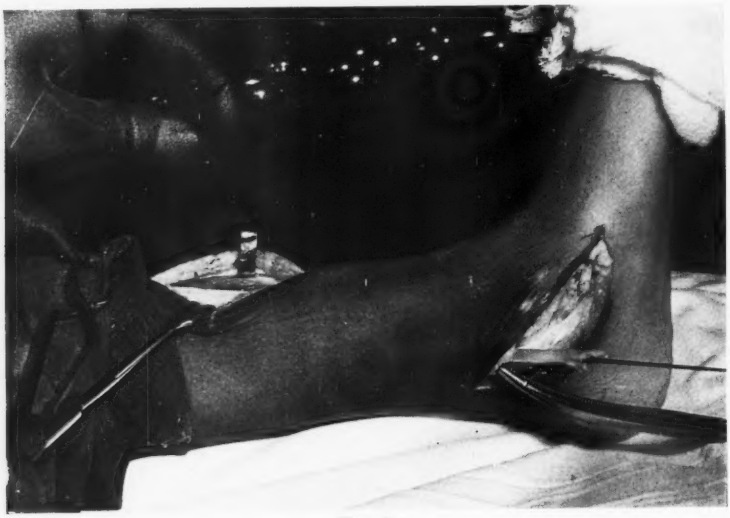


FIG. 1

Roberts' forceps passed proximally along the course of the tibialis posterior tendon hugging the tibia closely.

FIG. 2

The nose of the Roberts' forceps has been passed through the opening in the interosseous membrane and has grasped a double strand of strong thread.

opening the same length as the incision is made in it. A Roberts' forceps is introduced into the first incision and passed proximally, using the tendon of the tibialis posterior as a guide (Fig. 1). Hugging the tibia closely the forceps is pushed upwards through the aperture in

the interosseous membrane and a double strand of strong suture material is withdrawn into the first incision (Fig. 2). After tying this to the stay suture it is easy to withdraw the tibialis posterior through the interosseous membrane. This is much easier and less traumatic than trying to introduce a forceps from above downwards.

The first incision is closed and a third incision made over the dorso-lateral aspect of the foot. The tendon is drawn down subcutaneously into the third incision (Fig. 3). Before closing the second incision care must be taken to ensure that the part of the tibialis posterior muscle belly protruding through the opening in the interosseous membrane is not impinging on the sharp inelastic distal border. When necessary the gap in the interosseous membrane is enlarged distally to allow the transplanted tendon to glide freely.

In only one-third of the patients was it possible to secure the tendon to bone—either the lateral or the middle cuneiform—because the tendon was too short for safe anchorage. In the remainder the tendon was attached to the peroneus tertius tendon if this was present or to the lateral two tendons of extensor digitorum longus. This was done by the usual "buttonhole" method.

FIG. 3
The tibialis posterior tendon has been brought down to the dorso-lateral aspect of the foot for reinsertion.

After operation the transplant is protected for six weeks in a below-knee padded plaster with the foot in the over-corrected position. When the plaster is removed normal lace-up boots or shoes are worn. A strong plaster night splint is used for six months.

The view of Gunn and Molesworth (1957) that muscle re-education is easy after tibialis posterior transfer was confirmed in this series. No specialised retraining programme was adopted and no physiotherapists were employed for re-educative exercises. Many of the patients had active control of the transplant when the skin sutures were removed.

RESULTS

The criteria used to assess the results of tibialis anterior transfer (Singer and Fripp 1958) were again used in this series. A *relapse* was defined as the return of a foot to a state in which it was not plantigrade and either the adduction, inversion or equinus components of the deformity were not corrected to the neutral position. The assessment included the test for occult equinus (Singer and Fripp 1958).

The follow-up period varied from one year to three years. In twenty-seven of the twenty-eight feet operated on there was no relapse (Figs. 4 and 5). The shoes or boots wore evenly on soles and heels and no undesirable sequelae were observed except for a mild or moderate valgus of the hindfoot in four instances. This was possibly due to over-correction

FIG. 4
Bilateral congenital club feet twenty-one months after tibialis posterior transfer.

before operation. The amount of valgus has remained unchanged until the present. There was a relapse in one child due to the disinsertion of the tendon at its new attachment. The disinsertion may have been due partly to the fact that this patient was discharged from hospital after two weeks and only reappeared six months later walking barefooted. It was interesting that the relapse occurred in the direction of the adduction rather than the equinus component of the deformity.

FIG. 5
Negative test for occult equinus.

The operation of soft-tissue correction was performed in five feet, which are not included in this series, during the phase of varus wedging to correct the forefoot adduction. When the tibialis posterior tendons were exposed later all five were found unsuitable for transfer because they were thin, atrophic and adherent to the surrounding tissues.

Great care was taken to ensure that all three components of the deformity were completely corrected before the operation. This cardinal rule of tendon transference was relaxed in one instance and relapse occurred three months after the operation. This patient is not included in the series because the treatment began after this review had been completed.

SUMMARY

1. The tibialis posterior tendon was transferred in twenty-eight congenital club feet to maintain the correction obtained by serial wedge plasters.
2. There has been no relapse in twenty-seven of the twenty-eight feet in the period under review—namely, one to three years from operation.
3. The technique of the operation is described.
4. It appears that this operation should not be attempted when a soft-tissue correction has been done previously.

Since the completion of this paper Fried (1959) has reported thirteen patients on whom this operation has been performed. Twelve of the patients had results classified as excellent and satisfactory with a follow-up period of four to five years.

I have much pleasure in recording my thanks to my brother, Mr Alec Singer, for his constant encouragement and helpful criticism.

REFERENCES

BARR, J. S. (1958): Tendon Transplantation. *Journal of Bone and Joint Surgery*, **40–B,** 166.

BROCKMAN, E. P. (1930): *Congenital Club-foot*. Bristol: John Wright & Sons Ltd.

FRIED, A. (1959): Recurrent Congenital Club-Foot. *Journal of Bone and Joint Surgery*, **41–A,** 243.

FRIPP, A. T., and SINGER, M. (1953): The Kite Treatment of Congenital Talipes Equino-varus. *Postgraduate Medical Journal*, **29,** 391.

GUNN, D. R., and MOLESWORTH, B. D. (1957): The Use of Tibialis Posterior as a Dorsiflexor. *Journal of Bone and Joint Surgery*, **39–B,** 674.

SINGER, M., and FRIPP, A. T. (1958): Tibialis Anterior Transfer in Congenital Club Foot. *Journal of Bone and Joint Surgery*, **40–B,** 252.

RELAPSED CLUB FOOT

DILLWYN EVANS, CARDIFF, WALES

> " The literature on the treatment of club feet is, as a general rule, that of unvarying success. It is often as brilliant as an advertising sheet and yet in practice there is no lack of half-cured or relapsed cases, sufficient evidence that methods of cure are not universally understood."—E. H. Bradford at a meeting of the American Orthopaedic Association in 1889.

Congenital club foot is a variable three-dimensional deformity of a complex system of joints. Secondary and adaptive structural changes involving skeletal and soft tissues are already well established at birth and radiography is unhelpful in the first year of life. Our understanding of the subject is still incomplete, and when one reflects that it has to be based on dissections of rare post-mortem specimens, on radiological appearances in older children and on what can be seen through limited surgical incisions, it is not surprising that the literature is confusing and that inadequate observations gleaned from inadequate sources have led different observers to widely differing conclusions. After reading some of the literature one can only murmur with Sir Robert Jones when he was trying to summarise a vigorous discussion of the subject: " it only goes to show how many-sided the question is." It even seems probable that when, in the course of time, our knowledge does become complete we shall find that there is more than one explanation for congenital equino-varus and that " club foot " is a generic term covering several pathological entities.

This paper is not primarily concerned with the morbid anatomy of club foot. Its main purpose is to describe an operation which yields an adequate and stable correction of relapsed club feet. Experience of the operation suggests that it may have some theoretical as well as practical significance, for it is based on assumptions which appear to be justified by the results. The assumptions are that the essential deformity in club foot is in the mid-tarsal joint; that the other elements of the deformity, including varus deformity of the heel, are secondary and adaptive; and that full correction of the primary deformity combined with adequate release of all contracted soft tissues will yield a reasonably normal foot. The operation is simple and effective; in essence it merely adds wedge-resection of the calcaneo-cuboid joint to the already familiar procedure of medial and posterior release of contracted soft tissues. It appears to me to be a logical procedure, but before trying to establish the logic it is necessary to review the development of the hypothesis that the primary deformity is in the mid-tarsal joint—a hypothesis which is widely but by no means universally accepted.

PATHOLOGICAL ANATOMY

In 1803 Scarpa (Wishart 1818) pointed out that the deformity is essentially a dislocation of the tarsus on the talus and that " in infants . . . obliquity of the body of the astragalus is very trifling when compared with the other bones of the foot," and he predicted, correctly, that " descriptions of bones of the tarsus in congenital club foot will always differ from one another according to the degree of the deformity and the ages from which the descriptions are taken." In 1866 Adams, in a Jacksonian Prize Essay, noted that exposed parts of the articular surfaces of the talus which remain unengaged by the ankle and navicular bone were nevertheless covered by cartilage.

Thus Scarpa and Adams were clearly of the opinion that the talus was not at fault in this deformity, and although this view has since been repeatedly challenged, no reliable evidence has been produced to refute it. Elmslie, writing in 1920, accepted this view and went on to

say that the deformity was essentially one of the mid-tarsal joint. It was " a displacement of the navicular and cuboid inwards at the mid-tarsal joint with rotation of the os calcis which brings its anterior extremity downwards and inwards." He then added: " this position of the os calcis is simply an exaggeration of that which the bone assumes when the foot is plantar-flexed and inverted; the cuboid is subluxated inwards on the os calcis, the facet for articulation with it lies to the inner side of the anterior extremity of the os calcis . . . thus the anterior extremity of the os calcis becomes conical in shape." Ten years later Brockman (1930) came to the conclusion that the core of the deformity was the medial dislocation of the navicular on the talus—" a congenital subluxation of the head of the talus out of its socket, owing to a congenital atresia of the socket "—and that all other changes in the foot were adaptive.

These views are not universally accepted, but though there is some reason to doubt whether they describe and adequately explain all cases of congenital club foot, they do appear to describe satisfactorily most cases and to provide something on which a rational treatment can be based. They imply that the essential lesion in a club foot is a congenital dislocation of the navicular bone on the talus, that this bone carries with it the cuboid bone and the calcaneum, that changes of shape in the skeletal and soft tissues are secondary and adaptive, and that the essential element in manipulative reduction must be the replacement of the navicular bone on the end of the talus, so restoring the medial column of the foot. The operation for relapsed club foot which is described in this paper is based on these inferences and the effect of the operation appears to be evidence of their validity.

If we assume, therefore, for the purpose of this argument that the primary deformity is a subluxation of the talo-navicular joint and that the aim of treatment must be the reduction of this displacement, it becomes necessary to consider the part played by the secondary adaptive skeletal changes in maintaining this deformity and resisting its correction. The resistance encountered when a club foot is manipulated is essentially the resistance offered by any joint which has been dislocated for a long time—the resistance of contracted soft tissues and possibly of joint surfaces which are no longer congruous. But there is also in the lateral column of the foot a second line of resistance resulting partly from overgrowth in length of this lateral column and partly from the shape of the calcaneo-cuboid joint. The lateral column of the foot is not only longer than the medial column, being the arc of a bigger circle, but the shape of the calcaneo-cuboid joint offers its own resistance. Elmslie described the anterior end of the calcaneum as being " conical "; I cannot confirm this, but the anterior articular surface of the bone seems to face forwards and medially and not just forwards as in the normal. This adaptive change in the lateral column may not be very marked at birth, but it becomes a more important factor as the child grows older with the talo-navicular dislocation remaining unreduced, and it explains why the operation of medial release of soft tissues does not always result in a stable reduction of the deformity.

PRINCIPLES OF TREATMENT

The obstructive part played by the calcaneum and cuboid bone has long been suspected, and several attempts have been made to overcome it by shortening the lateral column. Ogston in 1902 enunciated " a new principle of curing club foot in severe cases in children a few years old." He advocated enucleation of the cuboid bone, of the anterior part of the calcaneum and of the head of the talus, but his method could not succeed because he ignored a first principle in the treatment of club foot—that the medial column of the foot must be restored and not weakened. More recently Johanning (1958) has revived the idea of enucleating the cuboid bone alone; it is clear, however, from his insistence on the importance of after-treatment and his admission that other operative procedures sometimes became necessary later, that although this may have helped to correct the deformity the correction remained unstable. Other methods have included wedge-resection of the cuboid bone, of the calcaneum,

of the mid-tarsal joint and of the tarsus as a whole, but all have failed to produce a correction of deformity which remained stable throughout the period of growth.

Clearly it is not enough merely to shorten the lateral column of the foot. It is also necessary to overcome the resistance offered by the obliquity of the calcaneo-cuboid joint and at the same time to facilitate reduction of the talo-navicular dislocation. This can be achieved only by wedge excision of the calcaneo-cuboid joint combined with release of the contracted tissues on the medial side of the foot to allow lateral shift and lateral rotation of the navicular bone. It has been found in practice that the operation does not always result in proper reduction of the talo-navicular dislocation, but it produces sufficient lateral rotation of the navicular bone to bring the first metatarsal bone into line with the talus, and the fused and shortened calcaneo-cuboid bone serves to hold it in this position (Figs. 1 and 2).

FIG. 1

FIG. 2

These sketches illustrate some of the effects of the operation on the skeleton. In some cases, especially in younger children, an accurate reduction of the talo-navicular dislocation is possible; in others, as in this sketch, the medial displacement of the navicular bone persists but sufficient lateral rotation of the bone is obtained to bring about a realignment of the tarsus.

The real test of the operation, however, and of the validity of the assumptions on which it is based, is whether it corrects the varus deformity of the heel. It does in fact do so, and as will be seen, it can even produce a gross valgus inclination of the heel. There is an evident relationship between the angle of inclination of the heel and the relative lengths of the medial and lateral borders of the foot, and the wider significance of this finding is discussed later.

THE OPERATION

The aim of treatment is to secure sufficient lateral rotation of the navicular bone to bring the first metatarsal bone into line with the talus, and to correct equinus.

The first step is to soften the foot and correct deformity as much as possible by means of the serial wedge plasters advocated by Kite (1935, 1939). Relapsed club feet are often tough and rigid, and better results are obtained from operations performed on pliable feet than on rigid feet.

The operation is performed through two incisions—a medial incision which allows division of contracted soft tissues, and a lateral incision which exposes the calcaneo-cuboid joint.

The medial incision starts just in front of the tubercle of the navicular bone and runs backwards along the line of the tendon of tibialis posterior, beneath the medial malleolus, and the

up the leg along the anterior border of the tendo calcaneus for a distance of two or three inches. The tight plantar structures are divided with a tenotome and any cavus which may be present can then be corrected with a wrench. The tendon of tibialis posterior is exposed throughout its length from medial malleolus to its insertion into the navicular bone. In some feet it will be found to be a well differentiated tendon; in others it may be poorly differentiated in its distal half inch and it may even merge with a small mass of tough fibrocartilaginous tissue which may occupy the space between the medial surface of the head of the talus and the medially subluxated navicular bone. The tendon must be dissected free of this slab of tissue and the tissue mass excised to expose the talo-navicular joint. The tendon is then lengthened by Z-plasty, taking care to allow for a substantial increase in length, and the capsule of the talo-navicular joint is divided on its superior, medial and inferior surfaces to allow free lateral movement of the navicular on the talus. The proximal half of the incision is then deepened to expose the tendo calcaneus, which is isolated and lengthened by Z-plasty. This will only occasionally allow correction of equinus; in most cases it is also necessary to divide freely the posterior capsule of the ankle and any tight strands of fibrous tissue that may be running parallel with the tendon; in some cases the tendon will be poorly differentiated, and very free dissection and division of tissue will be needed to free the back of the ankle and allow dorsiflexion of the joint. In a small minority of older feet even this free dissection will not permit correction to the right angle and the extra leverage of a wrench may be needed, but this wrench should not be used strongly and it should not be used at all until every strand of contracted soft tissue has been divided. It is, nevertheless, essential to correct equinus at least to the right angle before proceeding to the second part of the operation, and failure to do this at this stage will mar the final result. The elongated tendons should not be sutured until the second part of the operation has been completed.

The lateral incision crosses the calcaneo-cuboid joint and runs parallel with the tendon of peroneus brevis; the skin flaps are retracted and the joint is fully exposed. The next step, excision of the calcaneo-cuboid joint, is the critical stage of the operation. So far, nothing has been done except to make correction of deformity possible by releasing contracted tissues. Now by excision of a wedge of the right shape and size it is possible to correct any deformity that may be present. If there is cavus deformity the wedge should be thicker on its dorsal than its plantar surface and if the foot is rocker-shaped the wedge should be thicker on its plantar surface. If the forefoot is supinated the cuboid can be rotated on the calcaneum in such a way as to bring the head of the first metatarsal bone to the ground. If the correct wedge is removed the cut surfaces of the bones can be brought into apposition in such a way that all deformities can be seen to be corrected—and, incidentally, the navicular can be seen to have moved laterally on the head of the talus, thus exposing more of the medial surface of the head of this bone. It is important to realise that unless deformity is gross and the child old, the wedge will be thin—hardly thicker than the joint itself. It is easy to remove too much bone and to find that when the cut surfaces come together the deformity has been over-corrected. Those who have been accustomed to performing triple arthrodesis for talipes equino-varus need to be particularly careful because they may have been in the habit of over-correcting to allow for some relapse, especially in younger children. There will be no relapse after this operation because growth of the medial column of the foot will keep pace with growth of the lateral column and the shape of the foot will remain unchanged. It is therefore of the greatest importance to secure as exact a correction of deformity as is possible, and for this reason it is best to use two staples to hold the calcaneum and cuboid securely together; one staple is not enough because it will not prevent rotation of one bone on the other, and sutures are insecure and difficult to insert accurately.

The elongated tendons are then sutured and the wounds closed. The foot is immobilised in plaster until the joint is soundly arthrodesed. Radiological evidence of fusion is not seen in the young, and it is probably wise to continue immobilisation for about five months.

Walking in plaster is permitted after six weeks and ordinary shoes are worn after the plaster has been removed.

In twenty-six out of the thirty feet now under review the tendon of the tibialis anterior was transplanted to the outer side of the foot. This may have contributed something to the stability of the correction, but my impression is that it did not do so, and because it always produced a passive dropping of the forefoot, I have in recent years omitted it from the operation.

RESULTS

The operation was done on thirty feet between 1953 and 1956. The shortest follow-up period is therefore nearly four years and the longest nearly seven years. The ages of the children at the time of operation ranged from three years to fourteen years but most were between four and eight years.

It would be idle to attempt a statistical analysis of the results because the initial factors are too variable and the series is too small; one can only report impressions, and the first and most obvious impression is that the correction of deformity obtained at operation is, at any rate within the limits of the review period, permanent (Figs. 3 to 6). In no case has there been any change of shape in spite of the absence of any kind of after-care. If the shape is satisfactory when the plaster is removed, it will remain satisfactory. It is the constancy of this fact which, more than anything else, distinguishes this operation from any other performed by me for the relapsed club foot.

Another feature is that the operation can correct all elements of the deformity, including the varus deformity of the heel (Figs. 7 to 14). It is known, of course, that a soft-tissue release operation, alone and without calcaneo-cuboid fusion, can sometimes give the same correction in young infants and can even produce a rigid valgus foot if it is accompanied by an over-vigorous manipulation. However, the feet now under review are those of older children and it has become evident that even in this age group the subtalar joint retains a potential of mobility which is released when the mid-tarsal deformity is corrected. Sometimes, and usually in fibrotic feet, this potential is only enough to allow the heel to move into valgus; in others it is sufficient to yield a fairly mobile tarsus, and some of these feet have gained a surprising range of controlled side-to-side movement—more than enough to allow accommodation to sloping surfaces. The degree of mobility seems to depend on a number of factors: the age of the child at the time of operation (the best age is probably about six years), the severity of the deformity, the number of operations and manipulations already performed on the foot, and the elastic quality of its soft tissues.

A third impression is that the operation yields a serviceable foot of good shape, comfortable, strong and plantigrade. It allows the child to live a normal life free from such encumbrances as irons, altered shoes and night splints.

A less satisfactory feature is the limited active dorsiflexion and the tendency to dropping of the forefoot which results from transplantation of tibialis anterior to the outer side of the foot. This transplantation was omitted in four of the feet without ill effect, and these feet do not show the slight weakness of dorsiflexion which characterises the other twenty-six. I suspect that the transplantation is unnecessary and have omitted it from recent operations not included in this series, but it must be recorded as having been performed on twenty-six out of the thirty feet under review. It is a visibly functioning tendon in all of them, and it may therefore have been a factor in preventing relapse.

Technical errors account for six unsatisfactory results. In one the deformity was under-corrected, in two it was over-corrected and in three the result was marred by persistent equinus. Under-correction and over-correction result from lack of care when excising the calcaneo-cuboid joint. The wedge of bone which is excised must be of the right thickness; removal of insufficient bone will not fully correct the deformity and removal of too much bone

FIG. 3

FIG. 4

FIGS. 3 TO 6

Case 1—This was the first of the series. Both feet were clubbed at birth. The right foot was adequately corrected but the left relapsed into deformity. The operation was performed in June 1953 at the age of six. The radiographs (Figs. 3 and 4), which show the fused calcaneo-cuboid joint, and photographs (Figs. 5 and 6) were taken in November 1960.

FIG. 5

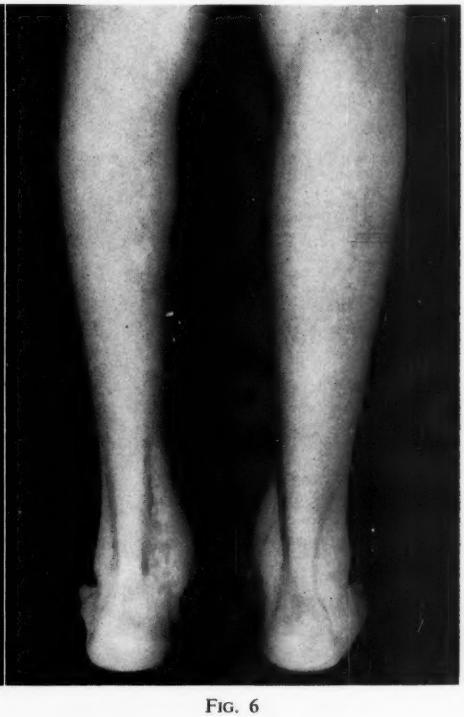


FIG. 6

FIG. 7

FIG. 8

Case 2—Feet before operation in a child aged five.

FIG. 9

FIG. 10

Case 2—Appearance of feet three years after operation.

will over-correct it (Figs. 15 to 17). The possible wider significance of this and the remedy for over-correction will be discussed later.

Persistence of equinus was at first thought to be due to adaptive broadening of the unengaged part of the upper articular surface of the talus, and corrective measures such as wedge osteotomy of the lower end of the tibia, section of the tibio-fibular ligaments and trimming of the talus were considered. Wedge osteotomy of the tibia was tried in two cases but with only a fair result in one and with a poor result in the other. Reconsideration of the problem in relation to the third foot suggested that persistent equinus might be due to nothing more than inadequate release of contracted soft tissues at the back of the ankle. The original operation was therefore repeated on this foot with greater thoroughness and an adequate correction was obtained. I am now persuaded by this, and by more recent experience, that broadening of the talus is not an important factor in preventing correction of deformity. It may sometimes prevent dorsiflexion above the right angle—which matters little—but it very rarely obstructs dorsiflexion to the right angle.

One more impression needs to be recorded. It is never wise to omit any part of this operation or to perform the operation in stages. Though performed through two incisions it is one procedure, and accurate correction of the deformity by excision of the calcaneo-cuboid joint is possible only while the tarsal joints are free to move into new positions—that is, while they are free from the restraining effect of soft tissues. This freedom from restraint is so important that it is never safe to omit any part of the release procedure, however unnecessary some part of it may appear to be.

DISCUSSION

The operation described in this paper serves two purposes—it provides a satisfactory method of dealing with the relapsed club foot and it yields experimental evidence of the validity of the theory that, whatever the etiology of the club foot may be, the essential deformity is a medial displacement or rotation of the navicular bone on the talus. Relapse results from recurrence of this displacement or, more probably, from failure to correct it fully at the primary manipulations.

This prompts some reflections on the primary treatment of club foot as well as on the management of relapsed and neglected cases. The conventional teaching on the treatment of club foot is that one should by manipulation " correct the adduction, inversion and equinus elements of the deformity," thus implying that there are three deformities that have to be corrected in turn; but it seems clear that such corrections, though at first apparently adequate, can be false and that their spurious character will only become apparent later when the foot is freed from its splintage and " relapse " occurs. It is unfortunate that late ossification of the tarsus deprives us of the help of radiography in these cases. Were the tarsal bones all ossified at birth, there seems little doubt that we would have learned to concentrate our efforts on repositioning the navicular bone on the talus, and that we would now be as alert to the danger of trying to correct adduction and inversion by applying pressure to the forepart of the medial border of the foot—thus causing the cuneiform joints to yield—as we are to the danger of correcting equinus by applying pressure to the forepart of the sole of the foot and so causing the mid-foot to yield and produce a " rocker " foot. The first step in the correction of a club foot should be replacement of the navicular bone on the talus; when this has been done the cuboid bone will have been pushed laterally and it will have taken with it the anterior end of the calcaneum which will have swung clear of the anterior end of the talus; the heel will have moved into valgus and the way will be clear for correction of equinus. Equinus is best corrected by pulling downwards on the back of the heel. Early tenotomy of the tendo calcaneus is sometimes practised and it has recently been suggested as a primary procedure, but one doubts the wisdom of this, for a tight calcaneal tendon can, by stabilising the heel, aid the reduction

FIG. 11

FIG. 12

Case 3—Feet before operation in a child of six.

FIG. 13

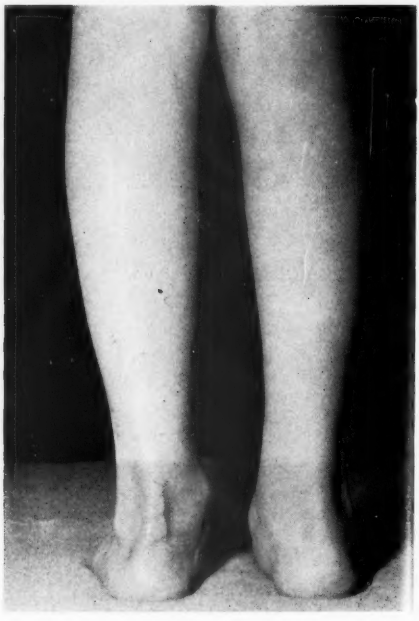


FIG. 14

Case 3—Appearance of feet three years after operation.

of the talo-navicular deformity and facilitate the lateral swing of the anterior end of the calcaneum, which in turn facilitates correction of the equinus.

It is not claimed that correct application of these principles will secure an adequate and permanent correction of every club foot. There are deformities which are so severe that, in my hands at any rate, they defy full correction. It is in such very resistant cases that early tenotomy of the tibialis posterior and calcaneal tendons might be thought to be helpful, but I have not found it so and I think it possible that in this small minority of exceedingly resistant cases relative overgrowth (or incompressibility) of the lateral column of the foot is already an obstructive factor, even at birth, and that one can do no more than maintain such feet in as good a shape as possible until surgical correction becomes feasible.

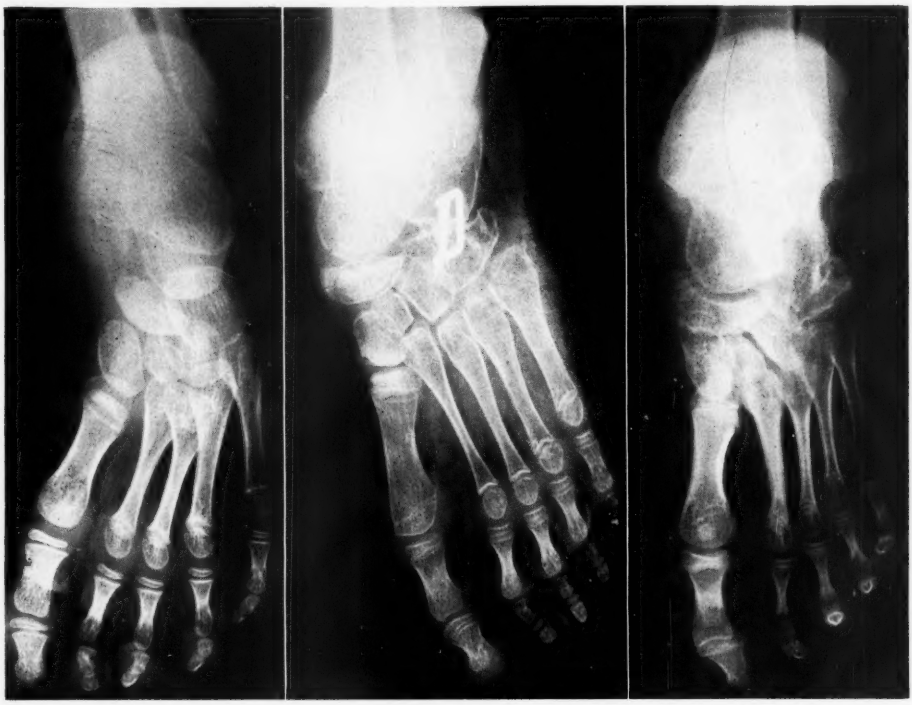


FIG. 15 FIG. 16 FIG. 17

Case 4—Here is shown the effect of over-correcting the deformity, and the changes that result from shortening and lengthening the lateral column of the foot. Figure 15—June 1955, before operation. Note the medial displacement of the navicular on the talus. Figure 16—April 1956, after wedge-resection of the calcaneo-cuboid joint. Too much bone has been removed and the talo-navicular dislocation is over-reduced. The clinical effect is a rigid valgus deformity of the foot. Figure 17—November 1960, after lengthening the calcaneum. The talo-navicular relationship is now normal and the clinical shape of the foot is satisfactory.

The improvement gained by manipulation has to be maintained at first by splintage and later by effective action of the peroneal muscles, but here again it must be kept in mind that even strong peroneal muscles cannot be expected to evert a dislocated foot and that it is of little use trying to develop peroneal activity when the navicular bone is still tucked around the medial side of the head of the talus.

If it becomes evident that the foot is relapsing into deformity treatment must start afresh but at this stage the resistance offered by the foot has become greater and full correction is often impossible. Mechanical aids are therefore employed and the traditional tool is a wrench or a wedge used under anaesthesia, but these can bruise the foot and lead to fibrosis of soft

tissue. Kite's method of correcting deformity by serial plasters is more gradual and probably more effective and less damaging to the tissues. It has justly found favour because it often yields satisfactory results; but it does not always succeed, and Kite (1957) has admitted that " if operative treatment of club foot, done at an early age, would give results as excellent as those obtained by operative treatment of other deformities, I would certainly support this approach."

This remark of Kite's echoes a widespread dissatisfaction with the effectiveness of the many operative procedures that have been suggested during the last sixty years for dealing with relapsed club foot and there can be little doubt that all have proved to be disappointing. Some have failed because they are unsound and others because they are inadequate. They can be divided into three broad groups—tendon transplantations, operations for release of contracted soft tissues, and bone resections or enucleations. The tendon transplantation operations are based on the assumption that the cause of relapse is muscle imbalance and, when this is so, they may succeed; but they must fail when the cause of the relapse is a dislocated joint and they therefore fail in most cases. Medial release of soft tissues fails when performed alone because, though soundly conceived, it is incomplete and does not take into account the obstruction offered by the deformed calcaneo-cuboid joint and the overgrown lateral column of the foot. It can correct the deformity in younger children but it does not stabilise the foot; relapse occurs unless the after-treatment is prolonged or the deformity is so over-corrected that a permanent valgus deformity is produced. The bone resections and enucleations have failed because they attacked either the medial column of the foot, such as the head of the talus or the talo-navicular joint, or the wrong part of the lateral column. It is obviously wrong to shorten or weaken the medial bony column of the foot; on the contrary, the aim should be to restore it and ensure that its growth in length keeps pace with that of the lateral column. The attacks on the lateral column are sounder in principle because they have been based on the obvious fact that this column, being relatively longer than the medial column, needs to be shortened, but they have failed in practice because it is not enough merely to shorten the lateral column by resecting a portion of the calcaneum or cuboid bone; it must be broken at a point that will permit sufficient lateral rotation of the navicular to bring the first metatarsal bone into line with the talus and it must be mended in such a way that it stabilises the foot by holding the navicular in its derotated position. This can be achieved only by excising and arthrodesing the calcaneo-cuboid joint.

Perhaps the most interesting single point that has emerged from this series of cases is the conversion of varus into valgus. In two of the cases the valgus produced was rigid and severe—a foot with a valgus heel and a convex medial border. The significance of this change was not at first appreciated and since it was obviously necessary to replace the heel beneath the ankle, a Dwyer-type osteotomy of the calcaneum was attempted but without success; the bone was freely divided but the hind fragment could not be moved and the deformity remained uncorrected. It then became obvious that excessive shortening of the lateral border of the foot had produced an excessive lateral rotation of the navicular on the head of the talus and that the remedy was to undo the calcaneo-cuboid fusion and to lengthen the lateral border of the foot by inserting a wedge graft (Figs. 16 and 17). This was tried and it produced, in varying degree, three effects; the heel moved towards neutral, the convexity on the medial border was reduced, and the foot as a whole moved slightly into equinus.

From this it appears that the relative lengths of the medial and lateral columns of the foot can be responsible, in some measure, for valgus and varus inclinations of the heel and that if these relative lengths are altered the inclination of the heel will change. Given a mobile or potentially mobile tarsus, the head of the talus will act as a pivot around which a short lateral column will pull the foot into valgus and a long lateral column will push it into varus. I am satisfied that this is so because it is a principle that has been seen to work not only in the correction of club foot and in the re-correction of the over-corrected club foot; it also works

in the correction of calcaneo-valgus. Calcaneo-valgus can be corrected by using a bone graft to lengthen the anterior part of the calcaneum; whether this correction is durable and worthwhile remains to be seen, but one point of relevance to this paper has emerged from experience of the operation; lengthening of the calcaneum produces two effects which are immediately visible at operation—it pushes the heel into varus and the ankle into equinus. It can, in fact, produce an equino-varus deformity of the foot and it thus provides some additional support for the view that the essential deformity in club foot is in the mid-tarsal region, and that the ankle and subtalar deformities are little more than inevitable consequences of this primary deformity. It does not however throw any light on the cause of this primary deformity, which remains a subject for speculation.

SUMMARY

1. An operation which can correct congenital club foot deformity in older children is described.

2. The operation is based on the assumption that the essential element in a complex tarsal deformity is a displacement of the navicular bone on the talus and that all other elements of the deformity are secondary and adaptive.

3. The results of the operation are noted and the implications of some of its effects are discussed.

I wish to thank Mrs J. Philip Jones of the Cardiff Royal Infirmary for the sketches, and I am grateful to Miss Wales and Mr Haddock of the Departments of Radiography and Clinical Photography of the Prince of Wales Hospital for their help.

REFERENCES

ADAMS, W. (1866): *Club-Foot*. London: John Churchill and Sons.

BROCKMAN, E. P. (1930): *Congenital Club-Foot*. Bristol: John Wright and Sons Ltd.

BROWNE, D. (1934): Talipes Equino-Varus. *Lancet*, **ii**, 969.

BROWNE, D. (1937): Modern Methods of Treatment of Club-Foot. *British Medical Journal*, **ii**, 570.

ELMSLIE, R. C. (1920): The Principles of Treatment of Congenital Talipes Equino-Varus. *Journal of Orthopaedic Surgery*, **2**, 669.

FRIED, A. (1959): Recurrent Congenital Club-Foot. *Journal of Bone and Joint Surgery*, **41–A**, 243.

FRIPP, A. T. (1951): The Relapsed Club Foot. *Proceedings of the Royal Society of Medicine* (Section of Orthopaedics), **44**, 873.

JOHANNING, K. (1958): Excochleatio Ossis Cuboidei in the Treatment of Pes Equino-varus. *Acta Orthopaedica Scandinavica*, **27**, 310.

KITE, J. H. (1932): The Treatment of Congenital Clubfeet. *Journal of the American Medical Association*, **99**, 1,156.

KITE, J. H. (1935): The Treatment of Congenital Club-Feet. *Surgery, Gynecology and Obstetrics*, **61**, 190.

KITE, J. H. (1939): Principles Involved in the Treatment of Congenital Club-Foot. *Journal of Bone and Joint Surgery*, **21**, 595.

KITE, J. H. (1957): Discussion American Academy of Orthopaedic Surgeons. *Journal of Bone and Joint Surgery*, **39–A**, 283.

OGSTON, A. (1902): A New Principle of Curing Club-Foot in Severe Cases in Children a Few Years Old. *British Medical Journal*, **i**, 1,524.

SINGER, M., and FRIPP, A. T. (1958): Tibialis Anterior Transfer in Congenital Club Foot. *Journal of Bone and Joint Surgery*, **40–B**, 252.

WILEY, A. M. (1959): Club Foot. *Journal of Bone and Joint Surgery*, **41–B**, 821.

WISHART, J. H. (1818): *A Memoir on the Congenital Club Feet of Children*. Translation from the Italian of Scarpa. Edinburgh: Constable and Company. London: Longman, Hurst, Rees, Orme and Brown.

A PROLONGED FOLLOW-UP OF PERONEAL SPASTIC FLAT FOOT

G. T. F. BRADDOCK, LONDON, ENGLAND

From the Royal National Orthopaedic Hospital, London and Stanmore

The subject of this paper is the natural history of peroneal spastic flat foot, and whether it has a radiographically demonstrable tarsal anomaly or not.

MATERIAL

The patients selected from the records were those who presented with symptoms of more than six months' duration between the ages of ten and fifteen years.

Treatment included one or more of the following: manipulation under anaesthesia, a below-knee walking plaster, or an iron and T-strap. At the time these patients were first seen

TABLE I

DETAILS OF TWENTY-EIGHT PATIENTS WITH PERONEAL SPASTIC FLAT FOOT

Number of patients examined:	
Men	24
Women	4
Total	28
Number of patients bilaterally affected	15
Number of feet causing symptoms:	
In adolescence	43
At time of review	24
Average period of observation in years	21
Longest	34
Shortest	13

TABLE II

RADIOGRAPHIC FINDINGS IN TWENTY-EIGHT PATIENTS WITH PERONEAL SPASTIC FLAT FOOT

Findings	Number of feet	Comment
Normal	27	17 feet caused symptoms in adolescence
Bony bars	22	19 feet caused symptoms in adolescence
Tarsal arthritis or gross beaking of talus	7	2 feet had talo-calcaneal bars, and 7 feet caused symptoms in adolescence

it was not the policy of the Royal National Orthopaedic Hospital to undertake early operation for this condition. It is unlikely, therefore, that any patients have " escaped " this series, of which details are given in Table I.

Four radiographs were taken of each foot: antero-posterior, oblique tarsal, lateral and the talo-calcaneal view (Harris and Beath 1948). No variation was made in the tube angle in either the talo-calcaneal or oblique tarsal views.

Radiographs which showed slight beaking of the talus only were included in the " normal " group in Table II. In all the feet with " bony bars " there was obvious radiographic evidence of a talo-calcaneal or calcaneo-navicular bar, complete or incomplete. In this simple classification, there is no separate mention of feet exhibiting minor tarsal anomalies, because there is no evidence that the latter are clinically significant.

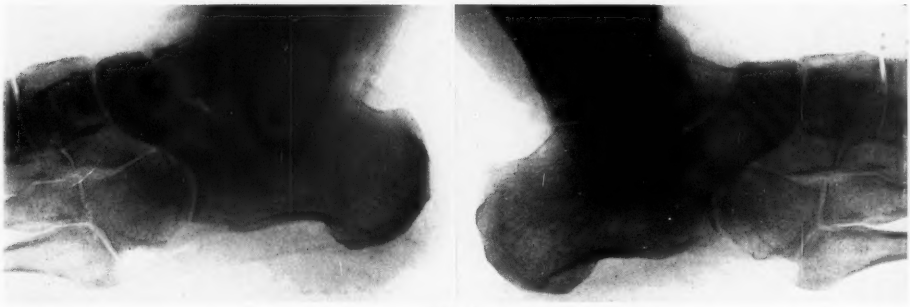


FIG. 1
Oblique tarsal views of both feet of a bus conductor aged forty-three. Bilateral complete calcaneo-navicular bars are shown but there is no tarsal arthritis.

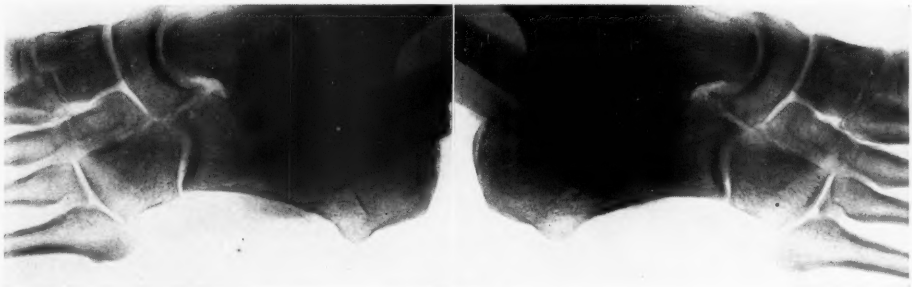


FIG. 2
Bilateral oblique tarsal views showing a relatively small head of talus in the presence of incomplete calcaneo-navicular bars.

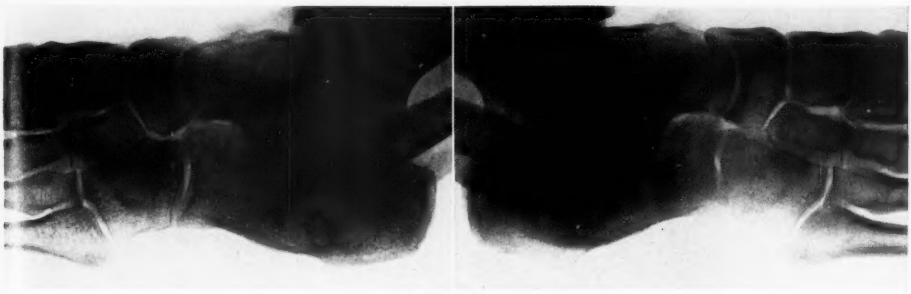


FIG. 3
Oblique tarsal views of normal feet for comparison with Figure 2.

Tarsal arthritis was not seen in any patient with a calcaneo-navicular anomaly. The oblique tarsal radiographs shown in Figure 1 are those of a bus conductor, aged forty-three years, who has complete calcaneo-navicular bars without tarsal arthritis. He was seen thirty-two years after his first attendance and complained of aching and stiffness in both feet only

after prolonged standing. This residual minor disability had remained unchanged, followin; a period of severe symptoms for eighteen months in adolescence.

The radiograph in Figure 2 illustrates the fairly constant association of a calcaneo navicular anomaly with a small head of the talus. Figure 3 shows oblique tarsal views o normal feet for comparison. Nine out of twelve radiographs with this anomaly showed thi feature.

One interesting feature is that severe symptoms were more persistent in patients withou obvious tarsal anomalies than in those with obvious bars (Table III).

The symptoms at the time of review are shown in Table IV. Patients with " mil symptoms " were not concerned in any way about their feet. All could walk two miles witl comfort; none had changed or selected his occupation because of his feet. The only complaint encountered were an ache in the foot at the end of the day or an occasional sharp pain afte

TABLE III

AVERAGE DURATION OF SYMPTOMS UNTIL COMPLETION OF TREATMENT

With bars .	18·2 months
Without bars .	26·3 months

TABLE IV

SYMPTOMS IN FIFTY-SIX FEET AT THE TIME OF REVIEW

Radiographic changes	Symptoms		
	None	Mild	Severe
Calcaneo-navicular bar	7	5	0
Talo-calcaneal bar .	2	6	2
Tarsal arthritis . .	4	1	2
Normal . . .	19	8	0
Total . .	32	20	4

TABLE V

LIMITATION OF INVERSION RELATED TO RADIOGRAPHIC FINDINGS IN FIFTY-SIX FEE

Radiographic findings	Greatly limited inversion	Total number in group
Calcaneo-navicular bar	8	12
Talo-calcaneal bar .	9	10
Tarsal arthritis . .	2	7
Normal . . .	4	27
Total . . .	23	56

twisting it. Patients with " severe symptoms " were concerned about their feet; they had selected work because of their disability and were anxious to have treatment. From Table IV it can be seen there is little relationship between the radiographic abnormality and the symptoms, certainly not enough to permit a clear prognosis in the individual patient.

All patients with residual symptoms were asked if there had been any change in their severity, and for how long they had been free of symptoms after their initial treatment. They said that they had always had some disability, and that this had persisted unchanged.

The relationship between limitation of inversion and the radiographic findings is shown in Table V. Patients with greatly limited inversion were unable to stand on the outer border of the affected foot. In four of the twelve feet with calcaneo-navicular bars there was a good range of inversion, and in three of the four the bony bar was complete.

The radiograph in Figure 4 is of the talo-calcaneal joints of a woman aged twenty-eight. There is a complete bony bar on the left; the right foot is normal. This patient had seve e symptoms in both feet at the age of ten years and wore an iron and T-strap on the left foot for three years. In spite of complete loss of inversion she has had no complaint since.

DISCUSSION

It is appreciated that this paper does not include those patients who first complained of pain in their feet during the third decade. The problem is to decide upon the treatment to offer the adolescent who may be unable to take part in vigorous school activities or start the career of his choice.

In favour of early tarsal arthrodesis it may be said that half the patients are likely to have minor symptoms for many years and perhaps for life, and that 10 per cent will eventually require operative treatment; also that the period of initial treatment is prolonged and largely symptomatic and probably has little if any permanent effect. A successful triple arthrodesis followed by three months in plaster would provide a more certain and more rapid relief of symptoms.

The arguments against early operation are that about half of the affected feet will regain full inversion and that slight symptoms or even persistent pain may be present after a triple arthrodesis.

FIG. 4

The talo-calcaneal joints of both feet of a woman aged twenty-eight years. A complete talo-calcaneal bar is present in the left foot. She had no symptoms after the age of thirteen.

SUMMARY AND CONCLUSIONS

1. The radiographic appearances are no guide to prognosis or treatment of peroneal spastic flat foot.
2. Only 10 per cent of peroneal spastic flat feet are likely to cause severe persistent disability.
3. Severe symptomatic tarsal arthritis is exceptional in this condition.

My sincere thanks are due to Mr H. J. Seddon for his help in the preparation of this paper. I also thank Mr R. Whitley of the Photographic Department of the Royal National Orthopaedic Hospital for the photographic reproductions.

REFERENCE

HARRIS, R. I., and BEATH, T. (1948): Etiology of Peroneal Spastic Flat Foot. *Journal of Bone and Joint Surgery*, **30–B,** 624.

THE CRUSHED CHEST

Management of the Flail Anterior Segment

W. SILLAR, GLASGOW, SCOTLAND

From the Southern General Hospital, Glasgow

The congestion of our roads and the higher speeds of motor vehicles have caused a progressive rise in the incidence of severe chest injuries and in consequence there is today a better appreciation of their lethal nature. The most outstanding landmark in a decade of progress has been the establishment by Carter and Giuseffi (1951) of the value of tracheostomy. By eliminating the dead space, facilitating the aspiration of secretions and reducing the resistance to air flow it enabled severely injured patients to survive. It led Blades (1955) to say that the need for traction was thereby eliminated except in the most severe cases. This may have retarded the search for a good method of internal fixation of the damaged thoracic cage, a tendency encouraged by the interest in the use of positive pressure respirators.

Although tracheostomy is undoubtedly the greatest single contribution to the problem it will not of itself ensure the survival of a patient with a large flapping anterior segment; and an efficient patient-triggered respirator, sensitive enough to eliminate flail movement, is still tantalisingly in the future. In our experience neither the Pask machine (Boyle, Gallie and Murray 1957) nor the Pneumotron (British Oxygen Company) nor the Blease " Pulmoflator " has been entirely successful in controlling flail movement. The method of inducing apnoea by hyperventilation with a simple positive pressure respirator advocated by Avery, Mörch, Head and Benson (1955) may solve the problem, but it has not yet been tried widely in this country.

In the meantime efforts to re-establish normal respiratory physiology by restoring the architecture of the thoracic cage are perhaps being abandoned too easily. This is especially true in anterior segment injuries. The poor condition of the patient, the desire to avoid any long operation and the technical difficulties of effective internal fixation of multiple rib and sternal fractures, have led surgeons to rely instead on procedures of proven value such as tracheostomy and simple traction and perhaps on positive pressure respiration. The penalties for failing to repair the anterior cage are, nevertheless, high, and when its repair is possible the benefits are immediate. It is the purpose here to illustrate the lethal nature of the anterior segment injury, to consider why this should be so and to show that there is a common type of anterior injury in which repair is easy and well worth while.

THE LETHAL NATURE OF THE ANTERIOR INJURY

The anterior injury is more severe because it reduces ventilation more seriously. It may be that it is also more likely to cause damage to the mediastinal viscera, and this may in some patients be the cause of death, but its ability to reduce vital capacity is much greater than that of the lateral injury, and this distinction is fundamental. If vital capacity is reduced below the critical level survival is impossible, and all too often this is the principal if not the sole cause of death.

In the four years 1955–58 records of twenty-three patients with flail thoracic segments admitted to Glasgow hospitals were traced and to these have since been added records of a further twelve patients. Of these thirty-five patients fifteen had injuries that were confined to the lateral part of the chest, eight had injuries involving only the anterior part of the chest, in four both aspects were clearly involved and in eight it was not possible to determine from the records the exact disposition of the flail portion (Table I). There were seven survivors

with purely lateral segment injuries and only one with a purely anterior segment injury. None of these patients was treated by rigid internal fixation of the sternum. The group excludes six patients to be described below who were treated by rigid internal fixation of the sternum.

TABLE I

MORTALITY IN THIRTY-FIVE PATIENTS WITH FLAIL SEGMENTS

	Number of patients	Deaths
Pure lateral segments .	15	8
Pure anterior segments	8	7
Mixed type . .	4	3
Details insufficient .	8	7

THE SHAPE OF THE ANTERIOR SEGMENT

In spite of the variability in the area, speed and direction of the damaging blow, and in the shape and elasticity of the chest before injury, there is a tendency for a common pattern to emerge, and it is possible to type the fracture as regards its effects and management in terms of one essential feature. In six patients seen at the Southern General Hospital a common pattern presented in five of them, and Figures 1, 2 and 3 are based on exact post-mortem studies in three of these. The essential common feature is the level of the sternal fracture, which has been at the second or third costal cartilage. Thus while the body of the sternum is in the flail segment, the manubrium is still part of the stable undamaged cage. The distribution of the rib fractures is less important, but the segment is always broader below than above. In most reports where details are given it is remarkable how frequently the same features are described and how often the upper limit of the flail segment has been the manubrio-sternal junction (Ghent 1959; Crastnopol, Stuckey and Kletschka 1957; Henry 1957; Daumet and Michel 1958).

ROLE OF THE STERNUM

In normal respiration four elements are described (Keith 1909, Campbell 1958). 1) *The lid of the thorax*, consisting of the first ribs and the manubrium, scarcely moves. In the classical anterior segment injury this part of the structure remains stable. 2) *The ribs* 2 *to* 5 swing outwards like bucket handles and upwards like pump handles to increase the capacity of the upper thorax. This function is affected to the extent to which these ribs are fractured and their anterior attachments are unstable. 3) *The diaphragm and the ribs* 6 *to* 10 *to which it is attached.* It is here that the disastrous effect of a flail sternum is apparent. Normally the diaphragm descends until it is stopped by the abdominal muscles acting through the viscera. It continues to contract and from this fulcrum the costal fibres now pull upwards on the lower ribs, which, being shaped like bucket handles, swing outwards. The normal action of the diaphragm is, therefore, firstly to descend, and secondly to swing the lower ribs upwards and outwards. By its descent the diaphragm is responsible for 60 per cent of the air exchanged. By descending one centimetre it pulls 270 millilitres of air into the normal chest. In anterior segment injuries the xiphisternum and most of the rib margin to which the diaphragm is attached are part of the flail segment, and in consequence the diaphragm cannot descend in a normal manner but glides ineffectually over the underlying viscera, pulling its anchorage backwards (Figs. 4 and 5). It also fails to increase the diameter of the lower thorax by swinging the lower ribs outwards because this action is dependent on an intact rib margin. 4) *The floating ribs* 11 *and* 12 *and the abdominal muscles*—These structures act together. They oppose the action of the diaphragm and limit its descent. This function is not affected by a simple anterior segment injury.

To this dire reduction of vital capacity must be added the paradoxical movement of the mobile segment which the anterior injury shares with the lateral injury. When all these factors are added together it is not surprising that, in spite of the enormous reserve, the tidal volume can often fall below the minimum essential for survival. The sternum is the key to it all, both directly and indirectly through its control of the movement of the rib margin.

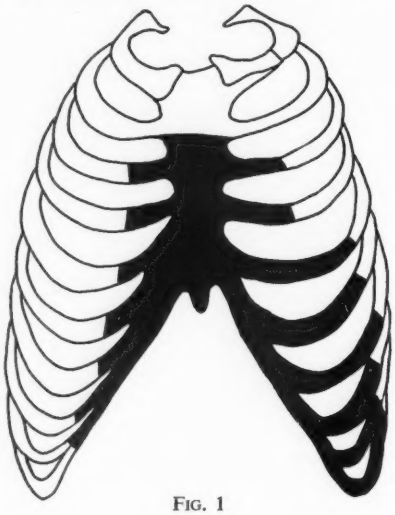


FIG. 1

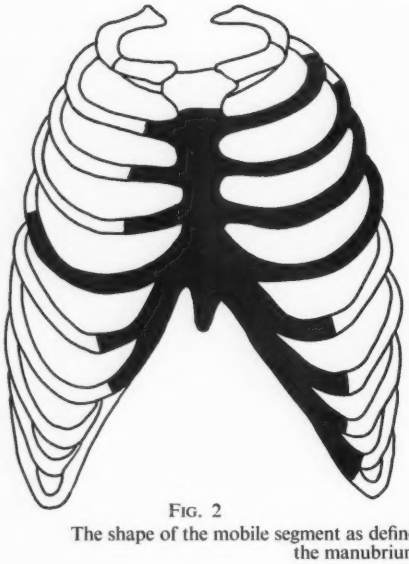


FIG. 2

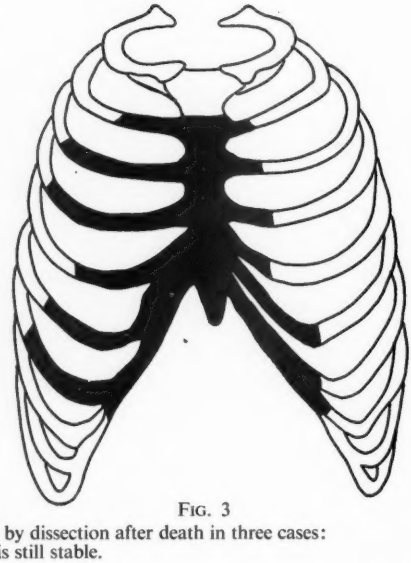


FIG. 3

The shape of the mobile segment as defined by dissection after death in three cases: the manubrium is still stable.

METHODS USED TO STABILISE THE FLAIL SEGMENT

Methods of controlling the segment can in general be divided into traction methods and internal fixation.

Traction methods—These include towel clips, wire loops, wire loops covered with polythene (d'Abreu 1958), cup hooks in the sternum (Rook 1951) and a Crutchfield skull caliper applied

to the sternum (Robin 1960). In general, traction has been by weights and pulleys. At the Birmingham Accident Hospital a bed frame with adjustable springs was used (Proctor and London 1955). To overcome the obvious difficulty inherent in the traction method of allowing for good coughing and postural drainage, a number of devices have been tried here, of which possibly the back brace and loop is the most feasible. But apart from the difficulties of the patient's posture the traction method has inherent faults; by its nature it must be either too strong or not strong enough. If it controls the worst of the paradoxical movement it simultaneously prevents good expiration. Good internal fixation, when it is possible, is better than traction if it restores normal respiratory mechanics.

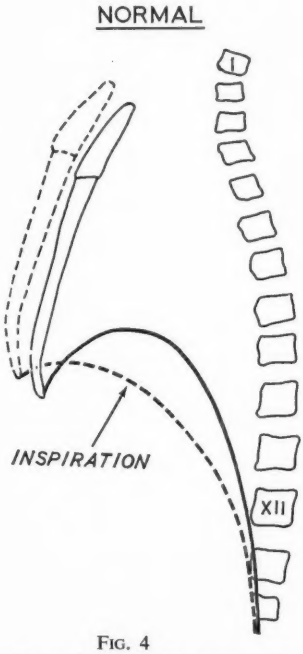


FIG. 4

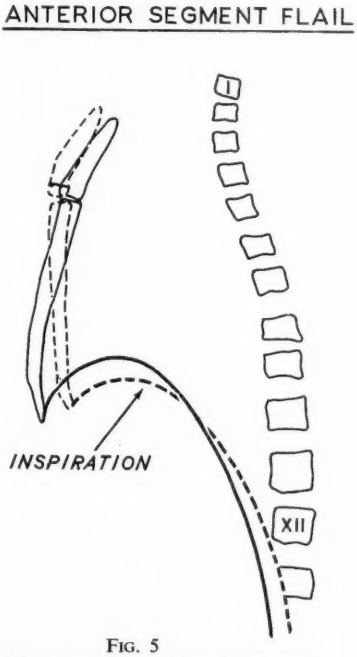


FIG. 5

Figure 4—As the diaphragm contracts the xiphisternum remains firm, the dome of the diaphragm descends and the thoracic volume is increased. Figure 5—As the diaphragm contracts the xiphisternum drifts, the diaphragm glides over the underlying viscera and fails to descend or to increase the thoracic volume effectively.

Internal fixation—Methods described include wiring rib ends together (Coleman and Coleman 1950, Bickford and Grant 1956), wiring fractures of the sternum, lashing adjacent ribs together, or Rush nails for medullary fixation of the fractured ribs at the apex of the mobile area (Crutcher and Nolen 1956), and the use of a plate bent to pass under the mobile ribs and support them (Jacques and Munro 1958).

Only one reference in the literature to a plate being applied to the sternum has been found. Henry (1957) described a classical anterior segment injury. He applied a six-hole plate to the sternum; the paradoxical movement of the damaged segment ceased and the patient survived.

RIGID INTERNAL FIXATION OF THE STERNUM

CASE REPORTS

Case 1—In September 1958 a man aged sixty-five was admitted to the Royal Infirmary, Edinburgh, after a road accident. He had a flail anterior thoracic segment. He was treated

by tracheostomy and intermittent positive pressure respiration. After eight days a Steinmann pin was passed upwards through the medulla of the mobile sternum into the stable manubrium; this controlled the paradoxical movement. The patient survived.

Case 2—In August 1959 a man aged fifty-eight was admitted to the Western Infirmary, Glasgow, after a lift shaft accident, with paradoxical movement of an anterior thoracic segment. The sternum was fractured at the level of the third rib. A plate was applied to the front of the sternum to control the paradoxical movement. Weight and pulley traction was also used, but this was later considered to be unnecessary. The patient survived.

Case 3—A man aged fifty was admitted to the Southern General Hospital in May 1958 with a flail thoracic segment involving the whole of the front and left side of the chest. He was treated by tracheostomy, but two days later he was still cyanosed and paradoxical movement persisted. A six-hole plate was applied to the sternum; paradoxical movement of the front and left side of the chest ceased at once and he was doing well until the eleventh day when he died suddenly from a coronary thrombosis.

Case 4—A man aged forty-eight was admitted to the Southern General Hospital in February 1960 with a compound fracture of the tibia and fibula and a flail anterior thoracic segment. The tibia was plated and a special sternal plate was used to immobilise the flail thoracic segment (Fig. 6). Tracheostomy was not performed. Recovery was uneventful.

Case 5—In May 1960 a man aged forty-two was admitted to the Western Infirmary with a head and chest injury which conformed to the classical anterior segment pattern. The special sternal plate was applied and tracheostomy was performed. Paradoxical movement was controlled and the patient's respirations were no longer distressed. He died on the second day. At necropsy death was shown to be due to cerebral damage. The lungs were clear. The chest wall was stable till the plate was removed from the sternum, when the flail movement of the damaged segment was again demonstrated.

If Henry's (1957) case is included we thus record six cases with the classical anterior segment injury, all showing paradoxical movement which was controlled by rigid fixation of the sternum. Two died, one from coronary thrombosis and one from cerebral damage.

THE STERNAL PLATE

Surgeons have, no doubt, been discouraged from plating the sternum because of its thinness and its awkward and variable shape (Fig. 7). A plate of 18-gauge stainless steel has been designed to fit its contours and to spread the load widely (Fig. 6). It can be adjusted by plate benders and there are eleven screw holes. The screws require to be half an inch, five-eighths of an inch or three-quarters of an inch long to engage both cortices (Fig. 8). Wood screws are preferable and the first bite of the thread should be accepted. A cuff may be fixed to the drill to prevent it penetrating too deeply. The internal mammary vessels are not in danger (Fig. 9).

If additional fixation of the mobile segment is desired a simple and effective method is by the insertion of Kirschner wires in the axis of the rib, traversing the fracture (Fig. 10). This procedure is not difficult provided there is no comminution, and it restores the continuity and rigidity of the rib without distortion. It can be used when the fracture is through the costal cartilage. The application of the sternal plate, with the addition of two or four lateral ties of Kirschner wire if required, is a simple surgical procedure (Fig. 11).

MANAGEMENT

In the present state of our knowledge any method of dealing with the problems of a crushed chest which has been described as useful should be considered. If the surgeon has a variety of procedures in mind, one or other of them may offer the best solution in a given case

Good methods of internal fixation, versatility in the application of traction, tracheostomy and intermittent positive pressure respiration must all be available and the choice made when all the factors are known.

FIG. 6

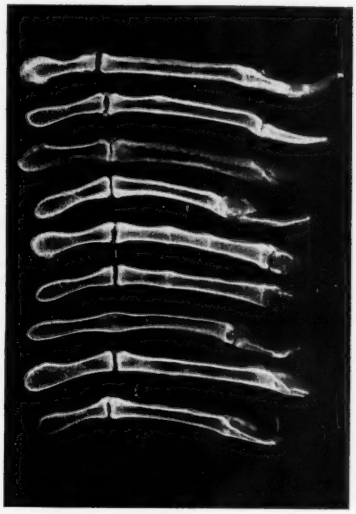


FIG. 7

Figure 6—The plate applied to the sternum in Case 4. Figure 7—The sternum is variable in size and shape. The cortical bone is thin and soft.

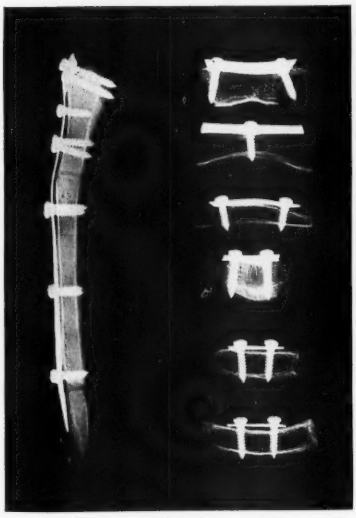


FIG. 8

FIG. 9

Figure 8—Screw lengths are one-half, five-eighths or three-quarters inch. Both cortices should be engaged and the first catch of the tightening thread should be accepted. Figure 9—The internal mammary vessels are not in danger.

A patient with a crushed chest is often desperately ill on admission and his survival depends on good emergency treatment. It may be necessary to take him at once to the theatre ante-room and anaesthetise him in order to provide ventilation by positive pressure before

there has been time to assess the skeletal damage. With positive pressure respiration the paradoxical movement disappears, and it may then be difficult to determine the nature of the skeletal defect and to choose the best method of dealing with it. After the patient's condition has improved, if a short acting muscle relaxant has been used the shape of the defect may

FIG. 10

A simple method of fixing rib fractures which does not damage the pleura is to pass a Kirschner wire along the axis of the rib across the fracture.

still be determined by allowing spontaneous respiration to return and then temporarily obstructing the airway. In the less urgent cases it will be possible to admit the patient to the ward and time will be available for fuller assessment. Pethilorphan 25–50 milligrams intravenously is of great value, and when this has taken effect it is time to examine the extent and nature of the damage of the chest wall. This is not easy. A large anterior segment can escape notice by its very size and because of the initial splinting of the chest wall by muscle spasm; and paradoxical movement is less obvious here than in the more localised lateral defects. Further, the nature of the movement of the chest wall can change radically with variations in the patency of the airway and the passing of the painful muscle spasm, so that repeated examination is necessary. Radiographs are essential for the recognition of pneumothorax and haemothorax and yield useful information about the damage to the skeleton, but they can be quite misleading about the extent of the injury. The correct choice of method will depend on correct appreciation of the skeletal damage. If the defect is an anterior one and the manubrium is still firm, the application of a sternal plate is indicated. Some lateral defects may lend themselves to fixation by the Kirschner wire method, and combinations of anterior and lateral damage may call for both. In some the damage is so extensive that the respiratory pattern defies description, and it is in these that tracheostomy and positive pressure respiration may offer the only hope. Here again, however, if a part of the picture is a recognisable flail segment its local fixation may make the difference between adequate and inadequate tidal exchange. Traction may still be an indispensable addition. Pleural drainage may be required. The advice and services of a thoracic surgeon may now be available, particularly for expert bronchoscopy, because tears of the main bronchi are easily missed. The decision to perform a tracheostomy will depend on the condition of the lungs, on the presence of a concomitant head injury and on whether tidal volume, when the patient is allowed to breathe spontaneously, is now adequate. In Case 4 tracheostomy was not necessary after a sternal plate had been applied.

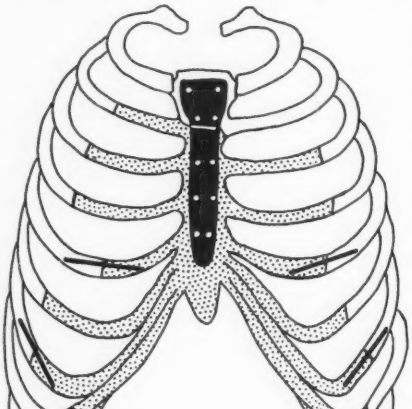


FIG. 11

The combination of a sternal plate and two or four lateral ties of Kirschner wire. To insert these is a simple operation that will stabilise some anterior segment injuries.

Tracheostomy should not be withheld if there is any doubt about the tidal volume. The patient's age and his cardiac and respiratory status before injury may influence the decision

SUMMARY

1. Anterior segment injuries are shown to be more dangerous than lateral segment injuries, and an explanation for this is offered.
2. A common pattern in anterior segment injuries is described.
3. When this pattern is present the application of a plate to the sternum is a useful procedure.
4. Six cases are quoted in which the application of a plate to the sternum was effective.
5. A plate has been designed for the purpose.

My thanks are due to Professor Roland Barnes for encouragement and to Mr Tom Howat, Mr Kenneth Frazer, Mr Ian Sinclair, Mr Henry Tankel and Mr John Leitch for permission to quote their cases. I am indebted to Mr D. N. McGuffie of Alexander Stephens and Sons, Shipbuilders, who made the first sternal plate, to Mr A. W. Laxen of the London Splint Company who now make it, to Dr J. M. Cameron for technical assistance, and to Mr Gabriel Donald for help with the illustrations.

REFERENCES

AVERY, E. E., MÖRCH, E. T., HEAD, J. R., and BENSON, D. W. (1955): Severe Crushing Injuries of the Chest. A New Method of Treatment with Continuous Hyperventilation. *Quarterly Bulletin of Northwestern University Medical School*, **29**, 301.

BICKFORD, B. J., and GRANT, A. F. (1956): Surgical Management of the "Stove-in" Chest Injury. *Annals of the Royal College of Surgeons of England*, **19**, 371.

BLADES, B. (1955): Management of Injuries to the Thorax. *Journal of the American Medical Association*, **159**, 419.

BOYLE, A. K., GALLIE, J. R., and MURRAY, D. B. (1957): Crush Injury of the Chest. A Report on Two Cases. *Anaesthesia*, **12**, 453.

CAMPBELL, E. J. Moran (1958): *The Respiratory Muscles and the Mechanics of Breathing*. London: Lloyd-Luke (Medical Books) Ltd.

CARTER, B. N., and GIUSEFFI, J. (1951): Tracheotomy, a Useful Procedure in Thoracic Surgery. *Journal of Thoracic Surgery*, **21**, 495.

COLEMAN, F. P., and COLEMAN, C. L. (1950): Fracture of Ribs—A Logical Treatment. *Surgery, Gynecology and Obstetrics*, **90**, 129.

CRASTNOPOL, P., STUCKEY, J., and KLETSCHKA, H. (1957): Management of Severe Crushing Injuries of the Chest. *New York State Journal of Medicine*, **57**, 2,379.

CRUTCHER, R. R., and NOLEN, T. M. (1956): Multiple Rib Fracture with Instability of Chest Wall. *Journal of Thoracic Surgery*, **32**, 15.

D'ABREU, A. L. (1958): *A Practice of Thoracic Surgery*. Second edition. London: Edward Arnold (Publishers) Ltd.

DAUMET, P., and MICHEL, J. (1958): Traumatisme thoracique avec volet antérieur traité par ostéosynthèse immédiate. *Poumon et le Coeur*, **14**, 219.

GHENT, W. R. (1959): Crush Injuries to the Chest. *Canadian Journal of Surgery*, **2**, 265.

HENRY, L. (1957): Case of Stove-in Chest Injury. *British Medical Journal*, **ii**, 339.

JACQUES, J. E., and MUNRO, W. D. (1958): Stove-in Chest. A New Method of Internal Fixation. *British Journal of Tuberculosis and Diseases of the Chest*, **52**, 160.

KEITH, A. (1909): The Mechanism of Respiration in Man. In *Further Advances in Physiology*. Edited by Leonard Hill. London: Edward Arnold.

PROCTOR, H., and LONDON, P. S. (1955): The Stove-in Chest with Paradoxical Respiration. *British Journal of Surgery*, **42**, 622.

ROBIN, G. C. (1960): Sternal Traction for the Flail Chest. *Lancet*, **i**, 26.

ROOK, J. R. (1951): The Value of Intravenous Procaine Analgesia in the Treatment of the Chest Injury. *Anaesthesia*, **6**, 221.

TIBIAL OSTEOTOMY FOR OSTEOARTHRITIS OF THE KNEE

J. P. JACKSON and W. WAUGH, MANSFIELD, ENGLAND

From the Harlow Wood Orthopaedic Hospital

It is generally agreed that an intertrochanteric osteotomy is a reliable operation for relieving pain in osteoarthritis of the hip (Blount 1952, Campbell and Jackson 1955, King 1957). The relief may be produced by a redistribution of weight-bearing stresses (Osborne and Fahrni 1950), by relief of tension in the stretched capsule and soft tissues (Lloyd-Roberts 1953) or by some unexplained vascular action. As early as 1936 Malkin pointed out that the correction of fixed deformity in an osteoarthritic hip was an essential part of the femoral osteotomy which he described. It was therefore considered that a corrective osteotomy might relieve pain in an osteoarthritic knee which had either valgus or varus deformity.

Surgical treatment of osteoarthritis of the knee is reserved for those patients who have failed to get adequate relief from physical treatment. If movement is severely restricted arthrodesis may be acceptable, and in some patients who have retained a reasonably mobile

FIG. 1

FIG. 2

Figure 1—Osteoarthritis of the left knee and genu valgum in a patient aged sixty. Figure 2—The same after upper tibial osteotomy.

joint, the scavenging type of operation is asserted to give satisfactory results (Magnuson 1946, Isserlin 1950). There are, however, a few patients whose osteoarthritis is associated with a lateral deformity and who have disabling pain together with a useful range of movement. The purpose of this paper is to show that in carefully selected cases osteotomy through the upper end of the tibia will relieve pain without stiffening the joint. We understand that this operation has been done in Liverpool and other centres but are not aware of any published results relating to its use in the treatment of the osteoarthritic knee.

CLINICAL MATERIAL

An upper tibial osteotomy was carried out in eleven patients in order to realign an osteoarthritic knee joint having significant lateral angulation. One of these patients died from

heart failure a year after the operation. This left for study ten patients upon whom eleven osteotomies had been performed. Four were men, six were women; their average age at the time of operation was sixty years. All these patients were seen and carefully questioned at

FIG. 3
Radiographs of an osteoarthritic knee associated with a valgus deformity, before (*left*) and eight months after operation.

FIG. 4
Radiographs of an osteoarthritic knee associated with a varus deformity, before (*left*) and nine months after operation.

varying periods after operation. The average length of follow-up was thirty-one months, the shortest being six months and the longest six years.

Indications for operation—All the patients suffered disabling pain unrelieved by conservative

treatment. Osteotomy was carried out when a valgus or varus deformity was present together with a useful range of knee flexion, preferably 90 degrees or beyond (Fig. 1).

Pre-operative lateral angulation—The deformity was valgus in seven and varus in four of the knees. The tibio-femoral angulation was measured in radiographs before operation. This angle was also measured in fifty healthy knees and found to average 2 degrees. In the seven knees with a valgus deformity that were operated on the angulation was 14, 14, 15, 16, 17, 21 and 22 degrees. In the four knees with varus deformity the angulation was 3, 5, 14 and 16 degrees. Figures 3 and 4 show the radiographic appearance before and after operation in typical valgus and varus knees.

Technique of operation—Various methods were used but a ball and socket type performed through cancellous bone at the level of the tibial tubercle was most satisfactory. The fibula is first divided through a separate incision over its middle third. The tibial osteotomy, convex either upwards or downwards, is outlined by a series of drill holes and completed with a gouge. The bone at this level is invariably soft and control of angulation is easy. Internal fixation is not necessary. The leg is immobilised after operation in a long plaster cast. The aim is to make the leg look straight (Fig. 2), and so the position is checked by post-operative radiographs; the plaster is wedged if necessary. Weight bearing is allowed in plaster after four to six weeks. Movement of the knee can usually be started after eight to ten weeks; recovery is remarkably rapid, 90 degrees of flexion usually being regained between two and six weeks from the time the plaster is removed.

RESULTS

Relief of pain—All patients say that their pain has been considerably if not completely relieved. When asked when the relief of pain was first noticed, several have commented that they were better immediately after the operation.

FIG. 5

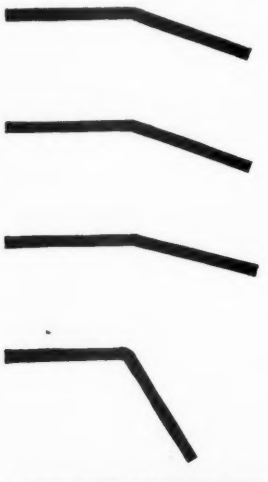


FIG. 6

Figure 5—Angle of flexion found at the time of review in eleven knees after upper tibial osteotomy.
Figure 6—Angle of greatest flexion in five knees after supracondylar osteotomy.

Recovery of movement—Figure 5 illustrates the angle of flexion found in the eleven knees at the time of review. Recovery was satisfactory except in one patient who had both knees operated on and who was immobilised for rather longer than is the present practice. In spite of restricted range of movement she has only occasional slight pain.

Radiographic changes—Radiographs taken before operation showed osteoarthritic changes of varying severity. There was invariably sclerosis in the femoral and tibial condyles on the side of the joint more subject to compression (the outer side in a valgus and the inner side in a varus knee). In one patient three and a half years and in another one and a half years after operation this sclerosis decreased, indicating that there had been an effective alteration in the mechanics of the joint. Apart from this there was no regression of the degenerative changes as measured by osteophyte formation and irregularity of the articular surfaces (Fig. 7).

COMPLICATIONS

Persistent instability—If the valgus deformity has been present for many years there may be considerable laxity of the medial ligament (Fig. 8). In one patient with such laxity the instability persisted after operation, but it is not so noticeable or troublesome now that the leg is straight. Extreme lateral instability might, however, be a contra-indication to the procedure.

Infection—In one patient a zig-zag type of osteotomy was followed by infection and sequestration. In spite of this the osteotomy healed, the knee regained 90 degrees of flexion and the patient has no pain.

Vascular complications—In no case was there any circulatory disturbance after operation. The danger of damage to the posterior tibial vessels must be appreciated and the osteotomy carried out carefully. It is felt, however, that correction of a lateral deformity is less likely to cause arterial damage or spasm than correction of a flexion deformity, when the vessels may be stretched.

DISCUSSION

If a severe lateral deformity of the knee persists from childhood, the abnormal transmission of weight-bearing forces through the articular surfaces may lead to osteoarthritic changes. In some of our cases, however, the angulation appears to be of relatively recent origin. It may therefore be possible that if osteoarthritis is confined to one or other compartment of the joint, angulation could be caused by loss of cartilage in that compartment and stretching of the capsule on the opposite side. Once the angulation has passed the stage at which the line of weight bearing falls well inside or outside the vertical axis of the limb, increasing deformity occurs for simple mechanical reasons. The study of this small group of cases has not solved this problem, and in some patients the deformity has certainly occurred first and osteoarthritis followed; but in others the sequence has been apparently reversed. Further observation on the natural progress of osteoarthritis is necessary before an answer can be given.

The site of pain in this type of knee also seems to vary and its cause may be different in different cases. The explanation may be mechanical, for it can be demonstrated radiographically that on weight bearing valgus angulation is increased (Fig. 8). Thus with every step pain could be caused by stretching of the soft tissues on the inner or open side of the joint. On the other hand some patients have pain on the opposite side of the joint, which is the site of compression on weight bearing.

Femoral osteotomy—In most knees with a valgus deformity radiographs show that the angulation is at the lower end of the femur. If an upper tibial osteotomy is carried out the leg can be made to look straight but the joint line remains tilted (Fig. 3). This theoretical disadvantage is offset by the ease with which flexion has been regained. There is no doubt either that the abnormal joint mechanics are improved and the angulatory stresses relieved (Fig. 7). Osteotomy through the femur may seem the correct procedure, but our experience with five such operations in four patients (Fig. 6) suggested that although relief of pain was good, residual stiffness of the knee was common. It was indeed this experience which led us to osteotomy through the tibia. When varus angulation occurs, mainly in the proximal end of the tibia (Fig. 4), there does not seem to be any theoretical contra-indication to osteotomy at this site.

FIG. 7

Radiographs of an osteoarthritic right knee associated with a valgus deformity, before (*left*) and three and a half years after (*right*) operation. Although there has been some increase in osteophyte formation and irregularity of the articular surfaces, there is less sclerosis in the lateral femoral and tibial condyles.

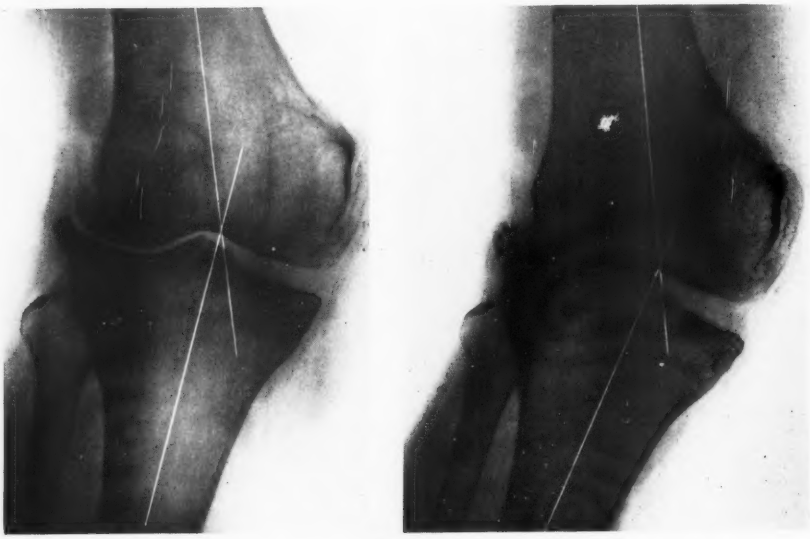


FIG. 8

Radiographs of an osteoarthritic right knee with the patient lying (*left*) and standing (*right*). On weight bearing the tibio-femoral angle is increased from 22 to 30 degrees.

At present it can only be suggested that the satisfactory results of tibial osteotomy are due in part to the alteration of the weight-bearing forces on the bone and in part to the reduction of strain on the stretched soft tissues. There is no doubt that our patients have greatly benefited from the operation, but many years must pass before its place in the surgery of the osteoarthritic knee can be finally assessed. The short term results do, however, indicate that upper tibial osteotomy is a justifiable and useful method of treatment provided the patients are carefully selected and the possible difficulties remembered.

SUMMARY

1. The results are presented of upper tibial osteotomy carried out in ten patients for osteoarthritis of the knee associated with lateral deformity.

2. The operation is indicated when there is severe pain, valgus or varus deformity, and a range of flexion of at least 90 degrees.

3. In every case pain has been relieved, and recovery of movement after operation has been easy.

We would like to thank Mr S. A. S. Malkin and Mr J. P. Campbell for allowing us to include some of their cases. We are also grateful to Miss H. M. Briggs, Research Secretary at the Harlow Wood Orthopaedic Hospital, for help in the preparation of this paper.

REFERENCES

BLOUNT, W. P. (1952): Proximal Osteotomies of the Femur. American Academy of Orthopaedic Surgeons. *Instructional Course Lectures*, **9**, 1.

CAMPBELL, J. P., and JACKSON, J. P. (1955): The Treatment of Osteoarthritis of the Hip by Osteotomy. *Journal of Bone and Joint Surgery*, **37–B**, 167.

ISSERLIN, B. (1950): Joint Debridement for Osteoarthritis of the Knee. *Journal of Bone and Joint Surgery*, **32–B**, 302.

KING, T. (1957): Proximal Femoral Osteotomy and Internal Fixation for Diseases and Injuries of the Hip. American Academy of Orthopaedic Surgeons. *Instructional Course Lectures*, **14**, 205.

LLOYD-ROBERTS, G. C. (1953): The Role of Capsular Changes in Osteoarthritis of the Hip Joint. *Journal of Bone and Joint Surgery*, **35–B**, 627.

MAGNUSON, P. B. (1946): Technique of Débridement of the Knee Joint for Arthritis. *Surgical Clinics of North America*, **26**, 249.

MALKIN, S. A. S. (1936): Femoral Osteotomy in Treatment of Osteoarthritis of the Hip. *British Medical Journal*, **i**, 304.

OSBORNE, G. V., and FAHRNI, W. H. (1950): Oblique Displacement Osteotomy for Osteoarthritis of the Hip Joint. *Journal of Bone and Joint Surgery*, **32–B**, 148.

THE ETIOLOGY OF CHONDROMALACIA PATELLAE

R. E. OUTERBRIDGE, NEW WESTMINSTER, BRITISH COLUMBIA, CANADA

A study of the literature on chondromalacia of the patella shows that its etiology is il understood. Most writers have been willing to accept injury as its cause without much question I have long been interested in this problem, and for some years I have been in the habit, wher excising a semilunar cartilage, of carefully observing and recording the condition of th articular cartilage on the deep surface of the patella. In 196 cases of medial meniscectomy th number of patellae showing healthy articular cartilage was approximately equal to the numbe showing pathological change (Table I). This is a lower incidence of chondromalacia tha that reported by many other writers. Wiles, Andrews and Devas (1956) stated tha chondromalacia patellae " often begins during the second decade, and by the age of thirt nearly everyone is affected." Øwre (1936) examined 124 patellae at necropsy and found a high incidence of abnormal cartilage: of subjects over twenty years of age, ninety-seven ou of 106 showed pathological changes.

Thus it must be accepted that chondromalacia of the patella is far more common than i generally believed, because usually it does not cause symptoms.

TABLE I

INCIDENCE OF CHONDROMALACIA OF THE PATELLA OBSERVED AT MENISCECTOMY

Age in years	Number of cases	Articular cartilage of patella	
		Normal	Abnormal
12–19	24	12	12
20–29	48	31	17
30–39	61	34	27
40–49	42	12	30
50–59	18	5	13
60–69	3	1	2
Total . .	196	95	101

MACROSCOPIC CHANGES IN CHONDROMALACIA PATELLAE

The normal appearance of healthy hyaline cartilage of the deep surface of the patella is bluish-white, smooth, glistening and resilient. The earliest change in chondromalacia is that the cartilage becomes dull or even slightly yellowish-white, and is soft and swollen. Characteristically, the site is almost always in the middle of the medial patellar facet, or just distal to that point, and is about half an inch or more in diameter (Fig. 1).

As the condition progresses, irregular deep fissures develop, and the affected area becomes a mass of villous-like cartilaginous flakes attached to subchondral bone (Fig. 2). This area gradually increases in size while at its centre the cartilage is eroded down to bare bone. Gradually the changes extend to the lateral facet, usually crossing the waist of the bone (Fig. 3) until the whole patella is affected.

The cartilage of the femoral condyles is not usually involved, with two exceptions. The first is the occasional " mirror " area of erosion usually seen in the intercondylar area (Fig. 4) in patients with advanced patellar changes or with generalised osteoarthritis of the knee;

and the second is the not infrequent erosion of the upper border of the medial femoral condyle, which will be discussed later.

These macroscopic changes of chondromalacia of the patella can be classified into four grades: in grade 1 there are softening and swelling of the cartilage; in grade 2 there are fragmentation and fissuring in an area half an inch or less in diameter; grade 3 is the same as

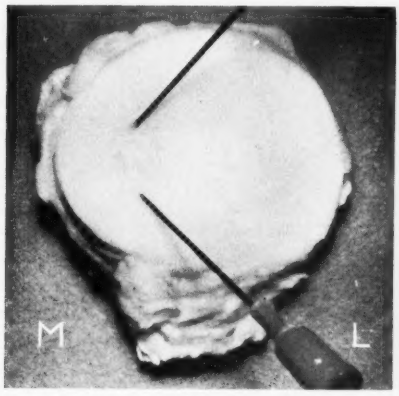


FIG. 1

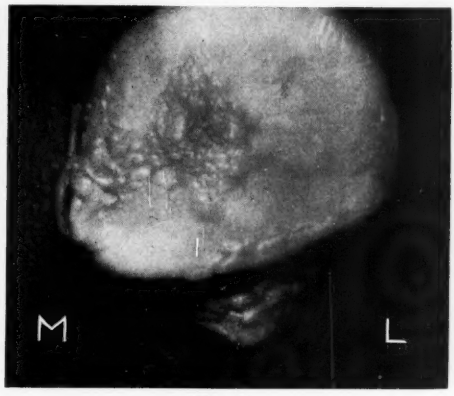


FIG. 2

Figure 1—The points of the needles show the edges of the chondromalacia at an early stage. Figure 2—The advanced fibrillar changes on the medial facet are spreading to the lateral side.

FIG. 3

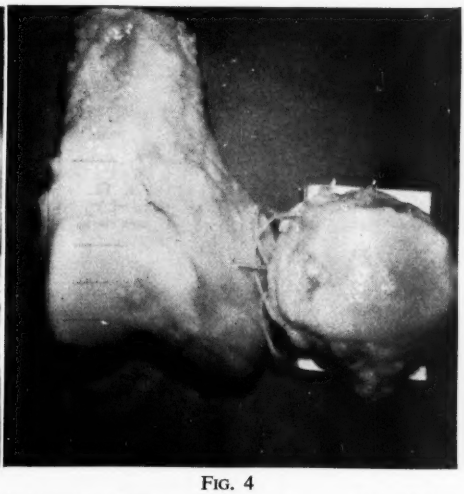


FIG. 4

Figure 3—The erosion is down to the subchondral layer medially, and has spread across the mid line. Figure 4—The medial facet of the patella is badly eroded, and there is a "mirror area" on the intercondylar area of the femoral condyles.

grade 2 but an area more than half an inch in diameter is involved; in grade 4 there is erosion of cartilage down to bone (Table II).

There appears to be no relationship between the time or severity of the injury causing the tear of the meniscus and chondromalacia of the patella. In this series there were seventy-eight "bucket handle" tears of the meniscus, and Table III shows that only thirty-seven of these were associated with chondromalacia of the patella, which was usually not severe.

THEORIES OF THE ETIOLOGY OF CHONDROMALACIA PATELLAE

In the literature various and inconclusive theories fall into three main groups.

Injury—This is the most popular theory. Some authors did not specify the type of injury (Axhausen 1919, Büdinger 1906), others suggested a direct injury (Chaklin 1939) or an indirect twist (Cave, Rowe and Yee 1945).

Generalised constitutional disturbance—An endocrine or toxaemic condition was suggested by Hinricsson (1939), but others believed that it was a combination of injury with an underlying constitutional tendency to cartilage degeneration (Kulowski 1933, Karlson 1940).

TABLE II

THE SEVERITY OF CHONDROMALACIA OF THE PATELLA SEEN AT MENISCECTOMY RELATED TO THE AGE OF THE PATIENT

Age in years	Number of cases	Grade of chondromalacia			
		1	2	3	4
12–19	12	8	1	2	1
20–29	17	6	6	2	3
30–39	27	7	12	6	2
40–49	30	9	13	6	2
50–59	13	3	5	3	2
60–69	2	—	1	—	1
Total . .	101	33	38	19	11

TABLE III

ASSOCIATION BETWEEN "BUCKET HANDLE" TEARS OF THE MENISCUS AND CHONDROMALACIA OF THE PATELLA

Age in years	Number of cases with bucket handle tears	Normal patellae	Grade of chondromalacia			
			1	2	3	4
12–19	9	5	3	1	—	—
20–29	22	15	2	2	2	1
30–39	29	17	4	6	2	—
40–49	14	3	3	6	1	1
50–59	3	—	2	1	—	—
60–69	1	1	—	—	—	—
Total . .	78	41	14	16	5	2

Patello-femoral contact—Wiberg (1941) investigated the congruity between the deep surface of patella and the femoral condyle at different angles of flexion, starting at 30 degrees of flexion. He found that the lateral patellar facet was concave in two planes and was in close contact with the lateral femoral condyle throughout most of the range of joint movement, but that the medial patellar facet was convex and only touched the medial femoral condyle with a small portion of its surface. He also found that contact between patella and femoral articulation is close at the beginning of flexion, but that this contact slowly passes outward

on to the medial and lateral facets of the patella with further flexion; in some specimens flexed beyond 90 degrees Wiberg observed that " incipient chondromalacia was seen precisely at the point of contact between the medial facet and the medial femoral condyle " which he felt was a proof that the stress over this small area of contact was likely to lead to chondromalacia.

Before any theory on the etiology of chondromalacia of the patella can be accepted, its great frequency, particularly after the age of thirty, and its common site of origin on the medial facet must be explained.

It is unlikely that any of the theories outlined above explains the etiology fully. Many patients, such as those reported in this paper, have no specific history of injury, nor does it seem possible that the mild and often repeated but quickly forgotten injuries to which the patella is vulnerable should be the cause of this condition, because other joints, such as the ankle, are subjected to far greater abuse without disintegrating. It also seems unlikely that a constitutional disturbance should make the medial patellar facet more susceptible to injury.

FIG. 5

FIG. 6

The quadriceps muscle is relaxed in Figure 5, but in Figure 6 the contracting quadriceps muscle has pulled the patella up and against the femoral shaft.

It has been suggested that the nutrition of the patellar articular cartilage, which is the thickest in the body, is at fault, but if this were so the degenerative changes should begin in the central ridge where the thickest cartilage is actually found (Øwre 1936, Wiberg 1941).

It is probable that the concept of pressure or friction in considerable flexion, as outlined by Wiberg (1941), comes closest to explaining the etiology, but flexion to this extent—such as in squatting—is not common in occidentals.

There is one other position, in the first 40 degrees of flexion, in which this particular part of the medial patellar facet is subjected to repeated pressure and friction. When the knee joint is fully extended the patella lies on the surface of the femoral shaft—from which it is separated by a subsynovial fat pad—and almost completely proximal to the crescentic superior border of the femoral articular cartilage. When the quadriceps muscle is relaxed the distal part of the patellar cartilage rests against this border (Fig. 5), but when the quadriceps is contracted the patella is pulled upwards about half an inch and compresses the fat pad and lies entirely over the femoral shaft (Fig. 6).

The crescentic upper border of the femoral articular cartilage extends more proximally on the lateral than on the medial side. Lying just above the lateral upper border, on the

femoral cortex, there is a smooth, whitish area, uncovered by soft tissue, which acts as a skid (Fig. 7) to allow the lateral patellar facet to glide smoothly on to the cartilaginous surface of the condyle; the lateral patellar facet is already well on to the condyle before the medial facet reaches the cartilaginous border of medial condyle.

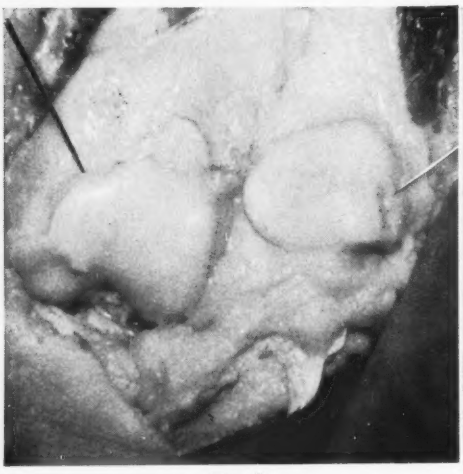


FIG. 7

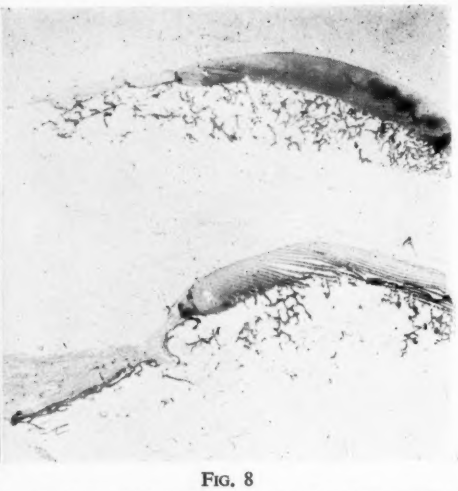


FIG. 8

FIGURE 7—The probe on the left is directed toward the rim of the thickened superior border of the medial femoral condyle. The " skid area " above the lateral condyle is well demonstrated. FIGURE 8—Longitudinal sections through the centre of the lateral (above) and medial (below) femoral condyles. Note the smooth continuity of the femoral shaft with the cartilage on the lateral side, and the rim on the medial side.

FIG. 9

FIG. 10

FIG. 11

Radiographs of the surface of medial condyle and medial patellar facet outlined with red lead to show their relationship. FIGURE 9—At 10 degrees of flexion. FIGURE 10—At 20 degrees. FIGURE 11—At 30 degrees.

The crescentic upper border of the medial condyle is quite different from that of the lateral. Here the articular cartilage is separated from the cortex of the anterior femoral shaft by a distinct rim which crosses the full width of the condyle and which may vary in height from three to eight millimetres (Fig. 8). As flexion begins, the patella, instead of sliding smoothly from the shaft on to the surface of medial condyle—as on the lateral side—particularly if the quadriceps muscle is contracted, at first hugs the femoral shaft closely, then

lifts up over the rim (Fig. 9). As flexion continues the patella is dragged across the rim to a point just beyond its centre (Fig. 10) and then tilts down on to the sloping surface of the condyle (Fig. 11). The part of the medial facet principally involved by this shearing force is this same area where the early changes of chondromalacia are seen. The range of movement through which this shearing occurs as the patella is drawn across the rim is from about 15 to 30 degrees of flexion.

This rim was present to a greater or lesser degree in most adult knee joints that were examined. Anatomists are aware of its presence, but it has never been given a name. In Figure 8 are shown longitudinal sections of the lateral and medial femoral condyles, to demonstrate the smooth approach of the former in contrast to the rim of the latter. Examination of knee joints at operation or necropsy has demonstrated that there appears to be a definite relation between the severity of the chondromalacia patellae and the size of this rim.

This theory of the etiology of chondromalacia of the patella has been presented because it is believed that it gives a satisfactory explanation of the pathological findings. However, there are probably other factors in the etiology as well as the size of the rim, such as the size and angulation of the medial, in relation to the lateral, patellar facet, and the power of the quadriceps muscle and its vastus medialis component.

SUMMARY

1. Chondromalacia of the patella starts most frequently on the medial facet.
2. The anatomy of the medial femoral condyle is described, including the rim at its superior border, and the different arrangement at the upper border of the lateral femoral condyle.
3. Rubbing of the medial patellar facet on the rim at the upper border of the medial femoral condyle can explain in part the etiology of chondromalacia.

I wish to acknowledge gratefully the help given to me by the Departments of Pathology and Radiology of the Royal Columbian Hospital, New Westminster, in preparing the material for this paper.

REFERENCES

ALEMAN, O. (1928): Chondromalacia Posttraumatica patellae. *Acta Chirurgica Scandinavica*, **63**, 149.
AXHAUSEN, G. (1919): Die Umschriebenen Knorpel-Knochenläsionen des Kniegelenks. *Berliner Klinische Wochenschrift*, **56**, 265.
BENNINGHOFF, A. (1925): Form und Bau der Gelenkknorpel in ihren Beziehungen zur Funktion. *Zeitschrift für Zellforschung und Mikroskopische Anatomie*, **2**, 783.
BÜDINGER, K. (1906): Über Ablösung von Gelenkteilen und verwandte Prozesse. *Deutsche Zeitschrift für Chirurgie*, **84**, 311.
CAVE, E. F., ROWE, C. R., and YEE, L. B. K. (1945): Chondromalacia of the Patella. *Surgery, Gynecology and Obstetrics*, **81**, 446.
CHAKLIN, V. D. (1939): Injuries to the Cartilages of the Patella and the Femoral Condyle. *Journal of Bone and Joint Surgery*, **21**, 133.
GRAY, C. (1948): Chondromalacia Patellae. *British Medical Journal*, **i**, 427.
HINRICSSON, H. (1939): Studies on Patellar Chondromalacia. An Attempt to Elucidate its Etiology. *Acta Orthopaedica Scandinavica*, **10**, 312–322.
HIRSCH, C. (1944): A Contribution to the Pathogenesis of Chondromalacia of the Patella. *Acta Chirurgica Scandinavica*, **90**, Supplementum 83.
KARLSON, S. (1940): Chondromalacia Patellae. *Acta Chirurgica Scandinavica*, **83**, 347.
KULOWSKI, J. (1933): Chondromalacia of the Patella. *Journal of the American Medical Association*, **100**, 1,837–1,840.
MATTHEWS, B. F. (1953): Composition of Articular Cartilage in Osteoarthritis. *British Medical Journal*, **ii**, 660.
OWRE, A. (1936): Chondromalacia Patellae. *Acta Chirurgica Scandinavica*, **77**, Supplementum 41.
OTO-HALL, R. (1945): Traumatic Degeneration of the Articular Cartilage of the Patella. *Journal of Bone and Joint Surgery*, **27**, 426.
VIBERG, G. (1941): Roentgenographic and Anatomic Studies on the Femoropatellar Joint. *Acta Orthopaedica Scandinavica*, **12**, 319.
VILES, P., ANDREWS, P. S., and DEVAS, M. B. (1956): Chondromalacia of the Patella. *Journal of Bone and Joint Surgery*, **38–B**, 95.

SUBCHONDRAL BONE CYSTS

C. G. WOODS, LEEDS, ENGLAND

From the Institute of Orthopaedics and the Royal National Orthopaedic Hospital, London and Stanmore

Cysts and pseudocysts in the subarticular bone adjacent to joints which are the site of rheumatoid arthritis or osteoarthritis are well known (Collins 1949, Plewes 1940). Less commonly, cysts arise in the same region of the bone when there is no disease of the joint. Review of the files of the Department of Morbid Anatomy of the Institute of Orthopaedics produced material from seven examples of such cysts, and it is the purpose of this paper to describe these lesions and discuss their possible pathogenesis.

CASE REPORTS

Case 1—A woman aged twenty-six complained of pain below the left knee after a fall from a cycle. She stated that she had sustained a similar accident at the age of sixteen and had suffered a little discomfort in the knee for a few weeks afterwards. Radiographs revealed two well defined cystic spaces with sclerotic bone margins in the posterior part of the upper end of the tibia. The joint surfaces and space appeared radiologically normal (Fig. 1). At operation the cysts were lined by a smooth grey membrane and contained a little gelatinous material. Treatment consisted in curettage alone and this produced complete relief of symptoms. Histological section of the tissue showed poorly cellular fibrous tissue with areas which had a hyaline appearance.

FIG. 1
Case 1—Antero-posterior radiograph of the knee.

Case 2—A woman aged thirty-nine complained of pain and swelling of the right wrist for three months after a sprain. Radiographic examination revealed a cystic lesion in the head of the ulna (Fig. 2). The affected area of bone was excised and the bisected specimen was seen to be a fibrous-walled cyst situated in the medullary cavity (Figs. 3 and 4). Microscopic examination showed that the wall of the cyst was composed of moderately cellular fibrous tissue in which there were small areas of mucoid degeneration. There did not appear to be a distinct lining layer of cells on the cyst wall. The bone trabeculae around the cyst were composed of living lamellar bone and were undergoing resorption in some areas, Howships lacunae and osteoclasts being present (Fig. 5). There were degenerative changes in the articular cartilage, but there was no evidence of complete loss of cartilage nor of a breach in the subchondral bone table.

Case 3—A man aged fifty-one had suffered from pain over the medial side of the right ankle joint for twelve years. Radiographs showed a clearly defined cyst with a thin sclerotic outline, situated in the medial malleolus (Fig. 6). The patient stated that ten years before a radiograph had also shown the cyst, but these films were not available for examination. He had not had any treatment after the first discovery of the cyst. The lesion was curetted and the cavity packed with bone chips. The graft incorporated and the symptoms were completely relieved. Histological section indicated that the curettings were from a multilocular cyst with a

FIG. 2
Case 2—Radiograph of the wrist.

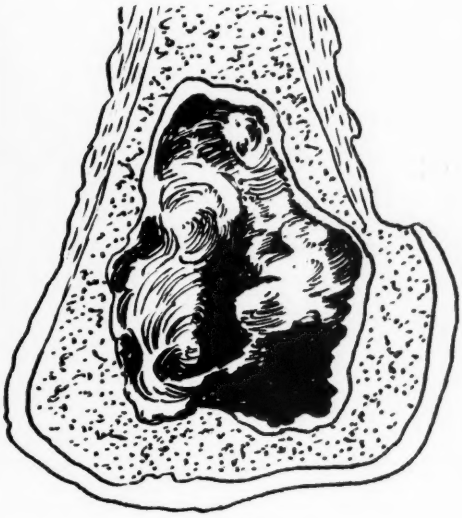


FIG. 3
Case 2—Drawing of bisected lower end of ulna. (×3.)

FIG. 4

FIG. 5

Case 2. Figure 4—Photomicrograph of cyst in the lower end of the ulna. (Haematoxylin and eosin, ×3.) Figure 5—Photomicrograph of the fibrous tissue of the cyst wall containing a few osteoclasts. Lamellar bone trabeculae with Howships lacunae show on the surface abutting on the cyst wall. (Haematoxylin and eosin, × 50.)

fibrous-tissue wall in which there were areas of mucoid degeneration and tiny cysts (Fig. 7). No distinct layer of cells was found on any of the surfaces, nor were osteoclasts seen. There was no bone in the specimen.

Case 4—A man aged thirty-six had had pain in his right ankle for about ten weeks before coming to hospital. He related the onset of the pain to a minor twisting injury. A clearly

FIG. 6
Case 3—Antero-posterior and lateral radiographs of the ankle.

FIG. 7
Case 3—Photomicrograph showing the oedematous fibrous tissue of the cyst wall in which " secondary " cysts are present. (Haematoxylin and eosin, ×110.)

defined cyst situated in the right medial malleolus was seen in the radiograph. The ankle joint appeared to be normal (Fig. 8). The lesion was curetted and packed with bone chips. Microscopic examination suggested that the cyst was multilocular with a poorly cellular fibrous tissue wall without a distinct lining layer of cells. Fibrinoid material was adherent to parts of the inner margin of the wall (Fig. 9). There was no bone in the specimen and no osteoclasts were found.

Case 5—A man aged forty-five complained of pain in the dorsum of the left foot of four months' duration. Radiological examination showed a well defined cyst in the lateral side of the lower end of the left tibia, with a normal ankle joint space (Fig. 10). At operation a double cyst, with a fibrous tissue wall and containing gelatinous fluid, was found and excised. For four years after operation the patient was symptom free, and then pain developed in the

FIG. 8
Case 4—Antero-posterior and lateral radiographs of the ankle.

FIG. 9
Case 4—Photomicrograph of part of the fibrous cyst wall with fragments of fibrinoid material adherent to the internal surface. (Haematoxylin and eosin, ×200.)

ankle joint. A radiograph now showed changes indicative of osteoarthritis. There was no evidence of the cyst. The wall of the cyst was composed of oedematous and poorly cellular fibrous tissue, and the contents of the cyst consisted of a loose fibrinous coagulum in the interstices of which was amorphous basophilic material (Fig. 11). There was no clear line of demarcation between the wall and contents of the cyst. Bone trabeculae around the cyst were part woven in construction (Fig. 12).

FIG. 10
Case 5—Antero-posterior and lateral radiographs of the ankle.

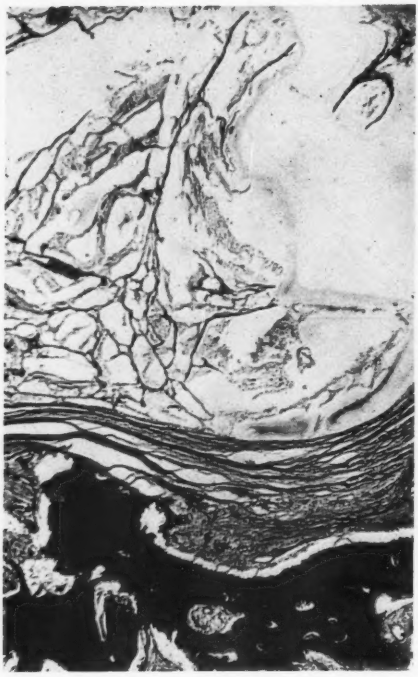


FIG. 11 FIG. 12

Case 5. Figure 11—Photomicrograph of the cyst showing a meshwork of fibrin to which mucoid material is adherent. The bone trabeculae outside the cyst are thickened. (Haematoxylin and eosin, × 15.) Figure 12—Photomicrograph showing a bone trabeculum abutting on the cyst wall which is composed of immature " woven " bone laid on the surface of mature bone. (Haematoxylin and eosin, × 180.)

Case 6—A man aged twenty-eight, after a twisting injury at football, suffered from pain in the right hip for about two and a half years. Radiographs showed a cystic lesion with a slightly sclerotic margin in the roof of the acetabulum. The hip joint showed some degree of subluxation but no osteoarthritic change (Fig. 13). The lesion was curetted and eight years later the hip joint still appeared radiologically normal, but calcification in the soft tissues around the joint interfered with joint function. The curettings consisted of fragments of sparsely cellular fibrous tissue in which there were several small circumscribed areas of mucoid degeneration.

Case 7—The material in this case consists of the head of the humerus removed at the necropsy on a forty-three-year-old achondroplastic male dwarf. Death was due to an astrocytoma and, so far as is known, no symptoms had been caused by this cyst. The cyst had a multilocular

FIG. 13
Case 6—Radiograph of the hip.

structure (Fig. 14), with a relatively thin fibrous tisue wall except in its most distal part where there was an irregular mass of fibrous tissue, poorly demarcated from the adjacent fibro-fatty tissue. Within this mass of tissue there were areas of oedema. The cavity had no distinct cellular lining and the contents were largely lost, only a little mucoid material and cellular debris remaining. Bone trabeculae around the cyst were of normal or increased thickness, of lamellar construction and entirely viable. No osteoclastic activity was seen. In many sections from the specimen discontinuity of the subchondral bone table was found at only one point (Fig. 15). The articular cartilage was intact throughout, the only abnormality being loss of basophilia in the matrix of the superficial zone.

CHARACTERISTICS OF THE LESION

The lesion occurs in young or middle-aged adults. Pain is the only presenting symptom and physical examination is either negative or reveals a little soft-tissue swelling over the affected area. Radiographic examination shows a well defined cyst, often outlined by a thin zone of bone sclerosis, situated close to the subchondral bone table. Histological examination of material from a curetted lesion shows thin strands of mature fibrous tissue of, at most,

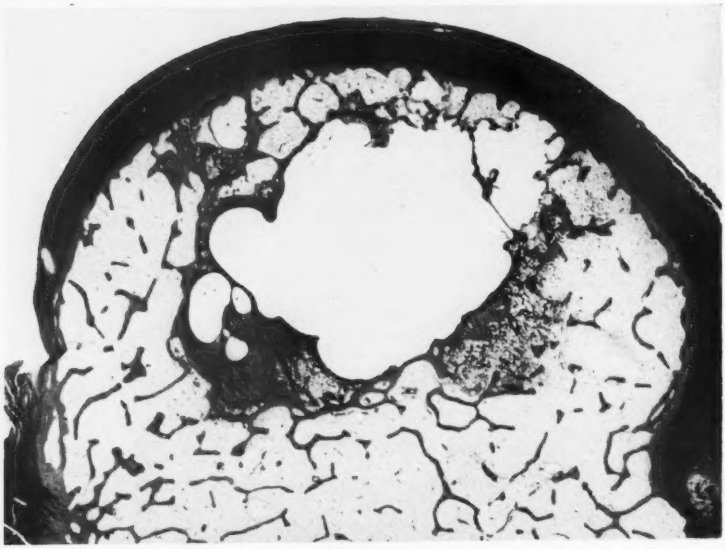


FIG. 14

Case 7—Photomicrograph of a section of the upper end of the humerus showing a complete cyst *in situ*. (Haematoxylin and eosin, ×5.)

FIG. 15

Case 7—A higher magnification of a section of Figure 14 to show the extension of the main cavity of the cyst up to the basal layer of the articular cartilage. (Haematoxylin and eosin, ×80.)

moderate cellularity, often containing areas of mucoid degeneration and even small " secondary " cysts. When seen intact the cyst may have a single cavity or be multilocular. The wall is relatively thin and there is no distinct cellular lining enclosing the cyst contents. Osteoclasts, when present, are confined to the peripheral part of the cyst, usually in relationship to bone. The fibrous tissue of the cyst wall does not display osteoblastic potentialities, the observed bone sclerosis being due to deposition of bone on the surfaces of pre-existing trabeculae with the production of either thick lamellar structures or compound structures, part lamellar and part woven bone. No evidence of bone necrosis was found. When cyst contents have been included in the histological sections they have been mainly of mucoid character with nuclear debris from necrotic cells. Eosinophilic fibrinoid material has occasionally been found. Curettage has been completely curative, whether followed by bone chip grafting or not.

DISCUSSION

The etiology and pathogenesis of these lesions is not clear. From the histological features, the site within the bone and the age distribution there can be little doubt that the cysts are distinct from such entities as non-osteogenic fibroma, simple bone cyst, aneurysmal bone cyst or a cystic form of fibrous dysplasia. The similarity between the fibrous tissue wall and the mucoid contents of these lesions and the appearances found in the material from " osteo-arthritic " cysts and some of the cysts associated with rheumatoid arthritis is striking. Although there is no unanimous view as to the etiology of the mucoid cysts in osteoarthritis and rheumatoid arthritis various suggestions have been made which merit consideration in relation to the pathogenesis of subchondral bone cysts. The theory of " blow out " of synovial fluid as propounded by Freund (1940) and Landells (1953) requires that a defect in the articular cartilage should exist. In the two specimens of this series in which the relationship between cyst and articular surface could be examined no portal for the entry of synovial fluid was found. Nor was there any evidence of fibrous or fibrocartilaginous repair of a temporary defect.

Replacement of bone trabeculae by inflammatory granulation tissue, as in rheumatoid arthritis (Collins 1949; Cruickshank, Macleod and Shearer 1954), can also be discounted in the lesions under discussion. Hicks (1956) reported four cases of " synovial cysts " in bone. His first two cases appear to be very similar to those described here. Hicks applied the term " synovial cyst " on the basis that the cyst lining is synovial tissue, and his suggestion about etiology was that the bone trabeculae are removed and the skeletal connective tissue undergoes synovial differentiation—a process of metaplasia. The interpretation that the lining of these cysts is true synovium is open to serious objection, in that there is no distinct lining layer of cells, nor the generous vascularity that is seen in synovium proper. In fact the counterpart of the cysts in bone are the ganglia and adventitious bursae in soft tissues. These last frequently have no direct connection with joint tissues and are, at most, "synovial-like." Furthermore, Collins (1949) suggested that they arise as a result of trauma on fibrous connective tissue. To talk of the formation of these cysts in bone as a process of metaplasia only provokes the question of what induces this deviation from normal in adult life. Rhaney and Lamb (1955) concluded that necrosis of bone played an essential part in the development of the cysts in osteoarthritis and produced convincing evidence of the existence of necrotic bone in their material.

Bugnion (1951) studied radiologically 600 wrist joints in adult cadavers. He found cysts in the small bones in 305 of the wrists, and in many of these there was no disease of adjacent joints. Eighteen of the lesions were submitted to histological examination, and from the description and photomicrographs it is clear that they are of the same nature as the lesions now under discussion. The cysts which were not associated with defects in the articular surface were considered by Bugnion to be " necrobiotic pseudocysts due to vascular disturbances." Although disturbances of the blood supply are described, none of the

photomicrographs in Bugnion's paper illustrated the developing stages; only the well developed lesions were portrayed. The hypothesis that these lesions arise as a result of localised necrosis of bone is attractive, but a careful search of my own material has failed to reveal any evidence of necrotic bone, nor was there any fibrosis of marrow outside the cyst itself. However, this may be because none of the material was removed from early lesions. Eggers, Evans, Butler and Blumel (1959*a*) reported the finding of subchondral cysts in the acetabulum of persons between the ages of twenty and forty years. It appears that such cysts can be asymptomatic for long periods and also that symptoms are often associated with the appearance of a sclerotic rim around the lesion. The same authors (1959*b*) also reported that lesions resembling those in patients may be produced in the femora and patellae of rats by immobilisation of the knee. The interpretation placed on their findings by these authors is that repetitive minor trauma to a localised area of bone results in cyst formation. It is obvious that the human material so far studied and reported in the literature has been obtained from well developed lesions. The acquisition of material from asymptomatic or poorly defined lesions is essential to test any theory that may be entertained with regard to the pathogenesis of subchondral bone cysts.

SUMMARY AND CONCLUSION

1. The clinical, radiological and pathological features in seven cases of subchondral bone cyst are described.
2. Various hypotheses on the etiology of the lesion are discussed.
3. Although the hypothesis of vascular disturbance is thought to be the most attractive one, it is concluded that study of material from much earlier lesions than that hitherto available is essential if any justifiable theory is to be propounded.

The author wishes to express his thanks to Dr H. A. Sissons for his help in this study and to the surgical staff of the Royal National Orthopaedic Hospital for access to the clinical notes on their patients. Thanks are also due to the technical staff of the Morbid Anatomy Department and to the staff of the Department of Photography.

REFERENCES

BUGNION, J. P. (1951): Lésions nouvelles du poignet. *Acta Radiologica*, Supplementum 90.

COLLINS, D. H. (1949): *The Pathology of Articular and Spinal Diseases*, pp. 186 and 71. London: Edward Arnold & Co.

CRUICKSHANK, B., MACLEOD, J. G., and SHEARER, W. S. (1954): Subarticular Pseudocysts in Rheumatoid Arthritis. *Journal of the Faculty of Radiologists*, **5**, 218.

EGGERS, G. W. N., EVANS, E. B., BUTLER, J. K., and BLUMEL, J. (1959*a*): The Pathogenesis, Significance, and Treatment of Subchondral Cysts of the Iliac Acetabulum. *Journal of Bone and Joint Surgery*, **41–A,** 766.

EGGERS, G. W. N., EVANS, E. B., BUTLER, J. K., and BLUMEL, J. (1959*b*): Subchondral Cysts: A Clinical and Experimental Study. *Journal of Bone and Joint Surgery*, **41–A,** 1,352.

FREUND, E. (1940): The Pathological Significance of Intra-articular Pressure. *Edinburgh Medical Journal*, **N.S. 47,** 192.

HICKS, J. D. (1956): Synovial Cysts in Bone. *Australian and New Zealand Journal of Surgery*, **26,** 138.

LANDELLS, J. W. (1953): The Bone Cysts of Osteoarthritis. *Journal of Bone and Joint Surgery*, **35–B,** 643.

PLEWES, L. W. (1940): Osteo-arthritis of the Hip. *British Journal of Surgery*, **27,** 682.

RHANEY, K., and LAMB, D. W. (1955): The Cysts of Osteoarthritis of the Hip. *Journal of Bone and Joint Surgery*, **37–B,** 663.

LONG SURVIVAL IN SOLITARY PLASMOCYTOMA OF BONE

C. J. E. WRIGHT, LEEDS, ENGLAND

From the Department of Pathology, University of Leeds

Although many examples of solitary plasmocytoma or myeloma of bone have been reported, in few has the diagnosis been clearly substantiated. Follow-up has often been too short because multiple myeloma, a closely related condition, may first manifest itself as a solitary growth identical with solitary plasmocytoma, and on occasion several years may elapse before other tumours appear. Sometimes the diagnosis of a solitary lesion has been made at necropsy, but even allowing for the difficulty in proving this, further tumours might still have appeared had the patient lived. Opinion has consequently hardened against the existence of true solitary plasmocytoma of bone, and prolonged survival after excision of the tumour, with failure to develop into multiple myeloma, seems to be the only reliable confirmatory criterion. The lack of such reports in the literature and their obvious importance in establishing the disease as an entity merits the recording of the following two examples. One is new; the other was reported from this department by Stewart and Taylor in 1932, but the after-history to date adds considerable support to the diagnosis.

Case 1—Man aged thirty-three. In December 1936 this patient, a labourer, slipped, suffering pain behind the right knee. The pain persisted for about three weeks, but no swelling was evident. He went back to work, but in February 1937 he again complained of pain behind the knee. It was aching in character, intermittent and worse on working, but there was still no swelling. The condition was treated as " rheumatism " until June, when he fell from a tar-spraying wagon and fractured his right femur. He was admitted to the Leeds General Infirmary under the care of Mr Broomhead. Radiographic examination showed a transradiant tumour in the middle third of the right femur, with a pathological fracture a little above the centre of the growth (Fig. 1). A biopsy specimen taken in August was reported by the late Professor Stewart to show a " typical plasmocytoma of great cellularity." The right humerus, radius and ulna, and the skull were radiographed but found to be normal. The tumour was treated by radiotherapy (3,025 r over thirty-seven days). A further radiograph a month later showed that the transradiant expansion of the shaft was more noticeable, but not extreme. The tumour then occupied six inches of the length of the shaft, and opaque trabeculae

FIG. 1

Case 1—Radiograph of mid-shaft of the right femur, showing expansive osteolytic growth with multicystic trabecular pattern.

ran irregularly through the transradiant area. Shortly afterwards, in November 1937, the leg was amputated by disarticulation through the hip.

Pathological examination—In the middle of the shaft of the femur there was a large new growth composed of compact greyish tissue which had been freely ploughed up by old and recent haemorrhages. The tumour had destroyed much of the cortex of the bone throughout the whole of the affected segment of the femur, but did not appear to be invading the adjacent muscles. Some evidence of regeneration of bone was present in places.

Microscopically, sections of the tumour stained with haematoxylin and eosin, and with Unna Pappenheim stain for plasma cells, showed a typical plasmocytoma (Fig. 2). The section contained closely packed well differentiated plasma cells. The cells were notably uniform, with moderate pleomorphism, and occasional immature forms; larger cells with two or three nuclei were evident. Mitotic figures were encountered, but only in small numbers. Parts of the tumour were unduly vascular, with a network of channels formed merely of endothelial lining cells. There were large areas of necrosis and haemorrhage. The tumour was

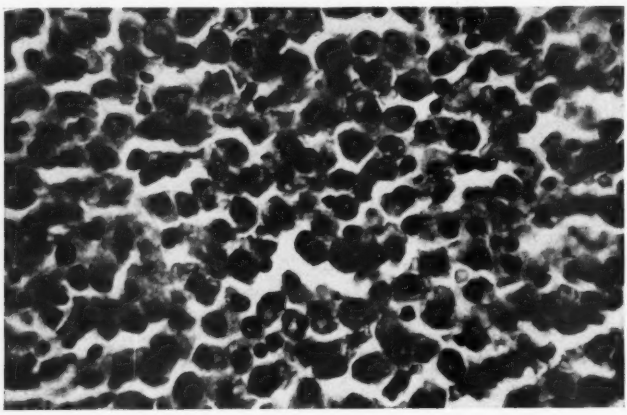


FIG. 2

Case 1—Plasma-cell structure of the tumour, with moderate pleomorphism. (Haematoxylin and eosin, ×650.)

separated from muscle by dense collagenous fibrous tissue in which slender spicules of bone were arranged at intervals along the margin. Inguinal lymph glands were found to be normal.

Subsequent progress—Since the operation the patient has shown no sign of recurrence, and when seen in June 1959, nearly twenty-two years later, he was in good health apart from persistent stomach symptoms. Partial gastrectomy was performed for gastric ulcer in 1955. Plasma protein estimations carried out recently on several occasions varied only between 5 and 6 grammes per cent. The blood picture remained normal.

Case 2—This case was recorded by Stewart and Taylor (their Case 1) in 1932 as a solitary plasmocytoma of the humerus, the diagnosis being supported by an eight-year survival after amputation. The patient, a farmer aged thirty-four, had a diffuse swelling of the upper part of the right arm. A radiograph had shown an endosteal growth at the upper end of the humerus, with expansion and thinning of the bone and a pathological fracture of its uppermost and middle thirds. A forequarter amputation was performed in 1924 and the humerus, which is now in the museum of the Institute of Pathology at Leeds, showed an irregularly lobulated tumour arising in, and largely destroying, the uppermost third of the shaft of the humerus (Fig. 3). The tumour was still contained within a thinned shell of bone except on the medial aspect where it had grown out into the adjacent soft parts. A full account of the histological

structure was given in the original case report, but the sections have been reviewed and the diagnosis of plasmocytoma has been confirmed. The tumour showed more pleomorphism than in Case 1, and there was invasion of muscle.

The original report stated that eight years had elapsed since the operation and that the patient was in perfect health. Jaffe (1958) cited a personal communication from Stewart informing him that this was still true eighteen years after the report. In July 1959 the writer visited the patient and found him perfectly fit apart from some osteoarthritis, and still farming, thirty-five years after the forequarter amputation.

FIG. 3
Case 2—Longitudinal section of solitary plasmocytoma of humerus reported by Stewart and Taylor in 1932. The specimen is in the museum of the Institute of Pathology at Leeds.

DISCUSSION

Plasmocytoma may be classified into three groups: 1) multiple myeloma or myelomatosis; 2) solitary myeloma or plasmocytoma of bone; 3) primary extramedullary myeloma or plasmocytoma. Multiple myeloma is uniformly fatal and usually of short duration, but occasionally there may be a relatively long survival. Kenny and Moloney (1956) reported survival in one case for nine years and in two cases for eleven years from onset of the disease in a series of fifty-seven cases. Other instances of even longer survival are on record. Multiple myeloma not infrequently presents initially as a single tumour, though the existence of a truly solitary form has been in some doubt. In a series of ninety cases of plasma cell myeloma, Carson, Ackerman and Maltby (1955) reported sixty patients in the multiple group, eighteen with an apparently solitary bone lesion, and twelve with primary extramedullary tumours. These authors believed that if followed for a long time the solitary bone lesion will eventually develop into diffuse myeloma. Of the cases in their series, one patient died within three months, seven survived from six to twenty-four months, and seven from two to six and a half years. Only three remained alive—after three years nine months, seven years four months, and nine years ten months. When a single tumour is the first manifestation of multiple myeloma, further tumours tend to appear rapidly, though on occasion there may be a delay of several years. In a patient originally described by Rogers (1930), later follow-up by Yentis (1956) revealed an interval of eight years from time of amputation. Yentis (1956) also reported, in his Case 3, an interval of ten years. No unequivocal case of solitary plasmocytoma of bone could be found by either Snapper, Turner and Moscovitz (1953) in their series of ninety-seven cases or Bayrd and Heck (1947) in eighty-three cases.

Although a number of examples of solitary plasmocytoma of bone have been recorded, and later accepted by other authors, there has been considerable uncertainty in recent years whether the tumour is in fact an entity, and, if so, what criteria should appertain for its

acceptability. The solitary growth is said to present as a large destructive lesion of bone usually with pathological fracture when a long bone is affected. According to Lumb and Prossor (1948) solitary tumours tend to be larger than any individual mass of the multiple disease, when the tumours are usually small and do not expand the bones. Snapper *et al* (1953), in ninety-seven cases of multiple myeloma, found palpable masses in only twenty-four. Raven and Willis suggested in 1949 that the tumour could be accepted as truly solitary only if no further tumours appeared and if careful radiographic examination of the rest of the skeleton remained negative for " two or three years or longer." Alternatively, they considered that adequate necropsy examination might prove the solitary nature of the growth. These authors accepted only eighteen cases, several of which were based on necropsy findings. As they pointed out, however, there could be no certainty in the latter instances that if the patient had survived other tumours would not have developed. Yentis (1956) emphasised that " the validity of a true solitary tumour would still pivot on meticulous and prolonged follow-up."

In two of the earlier cases accepted by subsequent authors recent information by Yentis (1956) has shown that they did in fact develop into multiple myeloma. Shaw's (1923) case was reported by Cutler, Buschke and Cantril (1936) to have survived for nine years, but Yentis (1956) was able to trace the later history and found that the patient had died from multiple myeloma with involvement of the spine and left femur. He also gave the after-history of the other case, in which the course proved to have been remarkably protracted. The patient had his right leg amputated in 1929 for a tumour described by Rogers (1930) as a solitary plasma-celled myeloma. All was well until 1937 when a further tumour was discovered in the left ischium, and a biopsy specimen showed a myeloma. In 1951 a destructive lesion was found in the left mandible and again a biopsy specimen revealed a myeloma. More masses appeared in the left temporal region and in the palate, and the patient died in 1953.

In contrast, additional follow-up information in two cases which similarly had been accepted lends support to the concept of a true solitary plasmocytoma of bone. Stewart and Taylor's case, herein further reported, is one instance. The other, that of Chesterman (1936), received added confirmation from a second report (Chesterman 1948) in which he stated that the patient was still well after twelve years. In a personal communication Chesterman has informed the writer that the patient died nearly four years later, or nearly sixteen years after amputation, from a cerebellar astrocytoma. Necropsy failed to show any evidence of plasmocytoma.

Two more examples of long survival have been reported by Christopherson and Miller (1950). In their Case 1 the patient was living sixteen years after hemimandibulectomy for solitary plasmocytoma of the mandible, and in their Case 2 a survival of twelve years after curettage and radiotherapy for a tumour of the uppermost third of the tibia was recorded. Yentis (1956), from personal communication with Christopherson in 1955, quoted later survival figures of twenty-two and nineteen years.

Notwithstanding these two instances Yentis believed that solitary plasmocytoma of bone, given a sufficiently long time, would eventually develop into multiple myeloma. Carson *et al.* (1955) were of the same opinion. Lichtenstein (1959) also thought that this was the " great likelihood " but on the basis of certain recorded cases with necropsy he admitted the possibility of a genuine solitary tumour. Jaffe (1958) considered that there were a few patients who, many years later, did not show myelomatous involvement of other bones. He specifically quoted the case of Stewart and Taylor. Raven and Willis (1949) believed that a truly solitary plasmocytoma of bone was an entity distinct from myelomatosis and that it could be cured by adequate local treatment. That all cases of solitary plasmocytoma of bone do not develop into multiple myeloma seems reasonably proven by a small group of cases which have been followed up for a long period after histological confirmation (Table I). Stewart and Taylor's case is outstanding in this respect, with a thirty-five year survival.

TABLE I

CASES OF SOLITARY PLASMOCYTOMA OF BONE WITH LONG SURVIVAL

Author	Age (*years*)	Sex	Site of tumour	Treatment	Survival after treatment (*years*)
Stewart and Taylor (1932)	34	M	Humerus: uppermost third	Amputation	35
Chesterman (1936) . .	39	M	Tibia: uppermost third	Amputation	16
Christopherson and Miller (1950)					
Case 1 . . .	29	F	Mandible: left half	Hemimandibulectomy	22
Case 2 . . .	25	F	Tibia: uppermost third	Curettage and radiotherapy	19
Wright (1961) . .	33	M	Femur: mid-shaft	Amputation	22

SUMMARY

1. A case of solitary plasmocytoma of bone with survival for twenty-two years after amputation is described.
2. Recent follow-up of a previous tumour reported from this department by Stewart and Taylor in 1932 has shown survival for thirty-five years after amputation.
3. These two instances of long survival, together with three culled from the literature, substantiate the hitherto doubted concept of a true solitary plasmocytoma of bone.

My thanks are due to Professor C. E. Lumsden for his helpful criticism of the manuscript, to Mr R. Broomhead for permission to publish Case 1, and to Mr J. T. Chesterman, of the City General Hospital, Sheffield, for the after-history of his case.

REFERENCES

BAYRD, E. D., and HECK, F. J. (1947): Multiple Myeloma. A Review of Eighty-three Proved Cases. *Journal of the American Medical Association*, **133,** 147.

CARSON, C. P., ACKERMAN, L. V., and MALTBY, J. D. (1955): Plasma Cell Myeloma. A Clinical, Pathologic and Roentgenologic Review of 90 Cases. *American Journal of Clinical Pathology*, **25,** 849.

CHESTERMAN, J. T. (1936): Solitary Plasmocytoma of Long Bones. *British Journal of Surgery*, **23,** 727.

CHESTERMAN, J. T. (1948): Solitary Plasmocytoma of the Long Bones. *British Journal of Surgery*, **35,** 440.

CHRISTOPHERSON, W. M., and MILLER, A. J. (1950): A Re-evaluation of Solitary Plasma-cell Myeloma of Bone. *Cancer*, **3,** 240.

CUTLER, M., BUSCHKE, F., and CANTRIL, S. T. (1936): The Course of Single Myeloma of Bone. *Surgery, Gynecology and Obstetrics*, **62,** 918.

JAFFE, H. L. (1958): *Tumors and Tumorous Conditions of the Bones and Joints*, p. 379. London: Henry Kimpton.

KENNY, J. J., and MOLONEY, W. C. (1956): Long-term Survival in Multiple Myeloma: Report of Three Cases. *Annals of Internal Medicine*, **45,** 950.

LICHTENSTEIN, L. (1959): *Bone Tumors.* Second edition, pp. 280–281. St Louis: The C. V. Mosby Company.

LUMB, G., and PROSSOR, T. M. (1948): Plasma Cell Tumours. *Journal of Bone and Joint Surgery*, **30–B,** 124.

RAVEN, R. W., and WILLIS, R. A. (1949): Solitary Plasmocytoma of the Spine. *Journal of Bone and Joint Surgery*, **31–B,** 369.

ROGERS, H. (1930): A Case of Solitary Plasma-celled Myeloma. *British Journal of Surgery*, **17,** 518.

SHAW, A. F. B. (1923): A Case of Plasma-cell Myeloma. *Journal of Pathology and Bacteriology*, **26,** 125.

SNAPPER, I., TURNER, L. B., and MOSCOVITZ, H. L. (1953): *Multiple Myeloma.* New York: Grune & Stratton.

STEWART, M. J., and TAYLOR, A. L. (1932): Observations on Solitary Plasmocytoma. *Journal of Pathology and Bacteriology*, **35,** 541.

YENTIS, I. (1956): The So-called Solitary Plasmocytoma of Bone. *Journal of the Faculty of Radiologists*, **8,** 132.

LOCKING OF THE METACARPO-PHALANGEAL JOINTS

J. W. GOODFELLOW, OXFORD and J. P. A. WEAVER, NEWCASTLE UPON TYNE, ENGLAND

From the Accident Service, Radcliffe Infirmary, Oxford

Derangement of the metacarpo-phalangeal joints of the fingers resulting in locking of the joints in flexion is not a well recognised condition. We have only been able to find five cases reported in the literature before 1960 (Gontermann 1928, Langenskiöld 1949, Alldred 1954, Flatt 1958). However, Aston has recently described five patients seen in one hospital during a period of ten months (Aston 1960). Our experience also suggests that the condition may not be so uncommon, therefore an accurate understanding of the mechanism of locking is of some importance.

The cases previously described show remarkable similarities to the cases reported here. A common cause for locking might reasonably be expected, and the purpose of this paper is to report five cases operated upon by the authors and to discuss the mechanism of the derangement, and the treatment.

CASE REPORTS

Case 1—A sixty-six-year-old man injured his right middle finger when pulling a suitcase off the luggage rack of a railway train. He thought the finger had been "bent backward," but immediately after the injury he found that he was unable to straighten it. On examination the following day the finger was held in 40 degrees of flexion at the metacarpo-phalangeal joint, and it was impossible to extend the joint actively or passively. Active flexion of the joint to 70 degrees was possible, and there was a full range of active movement in the other joints of the finger. Radiographs of the joint showed a small palmar osteophyte on the head of the metacarpal. Attempts to straighten the finger by force were unsuccessful but caused little discomfort to the patient. Manipulation under a general anaesthetic was attempted but an absolute block to extension was encountered. The metacarpo-phalangeal joint was therefore opened through a dorsal incision on its radial side. A small flake of articular cartilage lay free in the joint but had not caused the locking. In an attempt to discover a reason for the locking a curved dissector was thrust deeply, between the joint surfaces, towards the palmar metacarpo-phalangeal ligament. Immediately there was a "click" and the joint was free. When last seen eight months after the operation the finger had a full range of movement and there had been no recurrence.

FIG. 1
Radiographs in Case 2.

Case 2—A man aged fifty-one years was pulling a piece of wood which suddenly slipped and the middle finger of his left hand locked in flexion at the metacarpo-phalangeal joint. The physical signs were exactly similar to those described in the preceding case. Radiographs revealed osteophytes around the articular margin of the head of the third metacarpal (Fig. 1) and minor degenerative changes in the interphalangeal joints. Examination under general anaesthetic confirmed the presence of a complete block to extension which could not be overcome by manipulation. Through an incision similar to that described in Case 1 the joint was opened. There was evidence of degenerative arthritis, and the articular cartilage over the dome of the metacarpal head was deeply eroded, exposing the subchondral bone. Despite the insertion of a dissector into the joint and wide distraction of the joint surfaces the block to extension could not be overcome. The radial collateral ligament was then detached from the proximal phalanx and the inner aspect of the palmar metacarpo-phalangeal ligament was exposed.

Only when the latter ligament was incised, revealing the flexor tendons, did the joint suddenly snap free. When last seen four months after the operation, there was some residual swelling around the joint and limitation of flexion, no doubt the result of the extensive dissection and the degenerative changes. There was no recurrence of the locking.

Case 3—A woman aged sixty-five years lifted a bread bin and discovered that she could not straighten her left ring finger to disengage it from the handle. Five years earlier a similar locking of the same finger had occurred while she was pulling up a garden plant. On that occasion her doctor succeeded in straightening the finger by striking the back of her hand with a heavy book. When aged thirty-five years she had suffered an attack of "arthritis" affecting the joints of all her fingers and both knees, and she had suffered occasional pain in these joints since. We found a complete block to extension at the metacarpo-phalangeal joint as in the previous cases, and the radiographs showed degenerative changes of the small joints of the hand and osteophytes around the heads of the metacarpals (Fig. 2). Again manipulation under anaesthetic failed. Through a transverse incision in the distal palmar crease the tendon sheath in front of the metacarpo-phalangeal joint was exposed and opened. The structures were a little oedematous but no other abnormality of the flexor tendon or sheath was noted. Retraction of the tendons revealed the palmar metacarpo-phalangeal ligament, here forming the floor of the fibrous tendon sheath. A longitudinal incision through the dense transverse fibres of this ligament opened the joint and released the finger. It was possible to reproduce the locking by reapposing the cut margins of the ligament with the joint flexed. Attempted extension of the joint then caused the proximal edge of the ligament to "catch" at the head of the metacarpal, effectively locking the joint, which could again be freed by allowing the cut margins to part. The skin incision was repaired and movements of the joint were encouraged after operation. A full range of movement was rapidly regained and three months later there had been no recurrence.

FIG. 2
Radiographs in Case 3.

FIG. 3
Radiographs in Case 4.

Case 4—A woman aged eighty-three years locked the metacarpo-phalangeal joint of her right little finger while tugging at an obstinate knot on a parcel. On examination three weeks later the joint, though painless, remained locked in 45 degrees of flexion. Radiographs (Fig. 3) revealed osteoarthritic changes in the joints of her fingers and an oblique picture of the locked joint demonstrated the presence of a

hook-shaped osteophyte on the palmar aspect of the metacarpal head. Under local anaesthesia a transverse incision was made in the distal palmar skin crease, the flexor tendon sheath was incised and the tendons were displaced to reveal the palmar metacarpo-phalangeal ligament. There was a small sesamoid bone in the ligament just medial to the groove for the tendons. The " fan-shaped " fibres of the palmar metacarpo-phalangeal ligament (see below) were divided medial to the sesamoid bone and the joint unlocked. As the finger straightened a pointed osteophyte appeared between the divided fibres of the ligament. The locked state of the joint could be readily re-established, as in the previous case, by reapposing the cut margins of the ligament and thus re-establishing the tight ring in which the osteophyte had " caught." Before closure of the skin the osteophyte was trimmed off.

Case 5—A woman aged seventy-six caught her left middle finger while making a bed. She " felt something go " in the region of the metacarpo-phalangeal joint and was unable to extend her finger at that joint. She saw her doctor who diagnosed a sprained osteoarthritic joint and applied strapping. Extension was gradually regained but the finger was persistently painful and did not feel " right." Five weeks later her doctor referred her to hospital. On examination it was found that, whereas 20 degrees of hyperextension were possible at the other metacarpo-phalangeal joints, the middle finger extended only to the neutral position. Attempts to hyperextend the metacarpo-phalangeal joint produced pain referred to the palmar surface of the joint. An oblique radiograph revealed gross degenerative changes in the joint and a large hook-shaped osteophyte on the palmar surface of the metacarpal head (Fig. 4). The metacarpo-phalangeal joint was subluxated.

FIG. 4
Radiograph in Case 5.

It was clear that this woman's finger had locked in the same manner as those described in the previous cases. She had recovered almost full extension over a period of five weeks and operation was therefore not advised. Her complaints of pain in the joint and the feeling that something was out of place persisted and seven weeks after the original locking the joint was explored from the palmar surface under local anaesthesia as described in Case 4. The palmar metacarpo-phalangeal ligament was divided longitudinally, the incision being placed to the ulnar side of the flexor tendon sheath. The sheath was not opened. This manoeuvre immediately released the joint which could then be put into some degree of hyperextension. The osteophyte which presented through the incision in the ligament was not removed. The operation successfully relieved the symptoms and the patient said that the joint now felt normal. Three months afterwards the range of extension had been retained and, although grossly arthritic, the joint functioned as well as it had done before the episode of locking.

It seems likely that the gradual recovery of extension at this joint, which took place spontaneously over a period of six weeks before operation, occurred at the expense of subluxation of the joint. The primary cause of the locking remained unaltered, as demonstrated at operation. It seems unwise, therefore, to attempt treatment by " physiotherapy " which, like forceful manipulation, is unlikely to disengage the caught ligament and may result in further damage to the joint.

DISCUSSION

It was not until we had operated on the third of our patients that the mechanism of locking became apparent. The first case excluded the more obvious causes, such as a loose body or a fold of capsule interposed between the joint surfaces, and directed our attention to the palmar aspect of the joint. In the second patient a more extensive dissection showed that the collateral ligaments were normal and played no part in the maintenance of the locking, which persisted after the complete division of one of them. By a process of exclusion the palmar metacarpo-phalangeal ligament was strongly incriminated and its role was finally demonstrated in the third, fourth and fifth patients.

The anatomical text-books vary in their descriptions of the capsular ligaments of the metacarpo-phalangeal joints and the following account is based on dissections of fifteen fingers carried out by the authors.

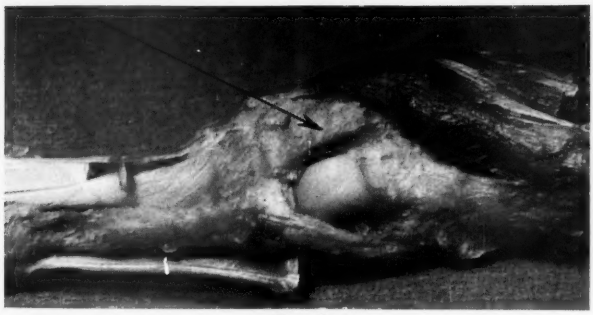


FIG. 5

Palmar aspect of metacarpo-phalangeal joint of a finger. The arrow points to the palmar plate, half of which has been excised to reveal its thickness.

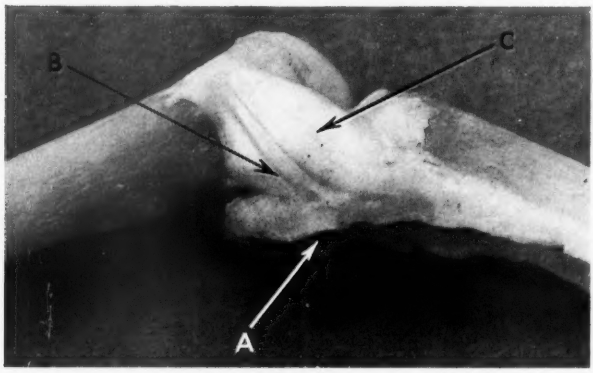


FIG. 6

Side view of dissected metacarpo-phalangeal joint of a finger. A—Flexor tendon sheath on the palmar aspect of the palmar plate. B—Fan-shaped ligament suspending the palmar plate from the side of the metacarpal head. C—Collateral ligament.

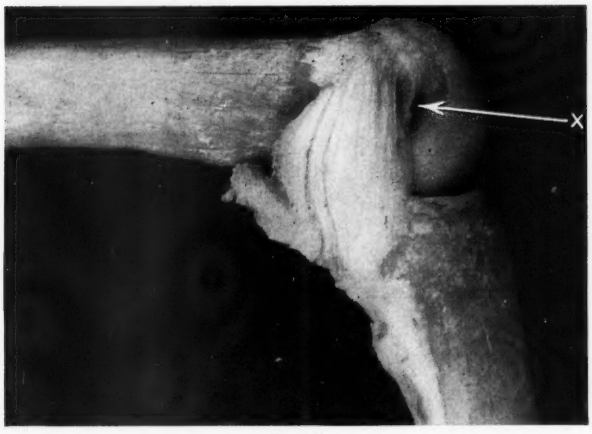
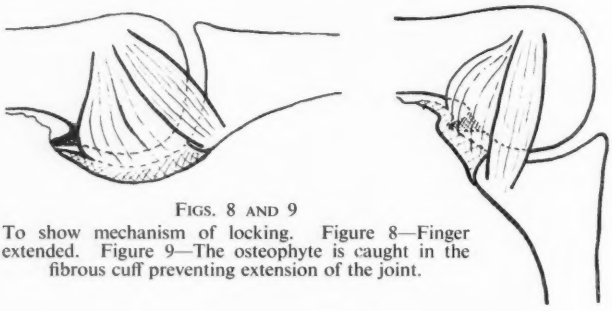


FIG. 7

Side view of dissected metacarpo-phalangeal joint in full flexion. The head of arrow shows the point at which an osteophyte may cause locking as supposed by Aston (1960).

The palmar metacarpo-phalangeal ligament is a thick fibrocartilaginous plate attached firmly to the base of the proximal phalanx but only loosely to the neck of the metacarpal (Fig. 5). In the index and little fingers it commonly contains a sesamoid bone (Joseph 1951). The fibres which comprise it run almost entirely transversely; those on its palmar surface turn forwards and encircle the flexor tendons to form the proximal part of the fibrous flexor tendon sheath (A in Fig. 6). Deeper, the fibre bundles of the deep transverse ligaments of the palm course uninterruptedly through the substance of the plate, uniting adjacent plates to one another. From a tubercle on the dorsum of the neck of the metacarpal there arise several thick bundles of fibres; the largest and most distally placed bundle forms the collateral ligament and is inserted into the base of the proximal phalanx (C in Fig. 6). More proximally two or three bundles of fibres fan out to become continuous with the deepest fibres of the



FIGS. 8 AND 9
To show mechanism of locking. Figure 8—Finger extended. Figure 9—The osteophyte is caught in the fibrous cuff preventing extension of the joint.

palmar plate along the whole length of the lateral margin of that structure (B in Fig. 6). This " fan-shaped " ligament is distinct both in its function and its mechanics from the collateral ligament proper. The latter structure maintains lateral stability of the joint, the former acts as a suspensory ligament for the palmar plate, and forms with it a fibrous cuff, which in full flexion glides off the articular surface of the head of the metacarpal and comes to lie around the neck of the bone (Figs. 6 and 7).

Mechanism of locking—Figures 8 and 9 represent diagrammatically how an osteophyte situated on the palmar articular margin of the head of the metacarpal may catch in the " fibrous cuff " described above and, through the medium of the palmar plate, prevent extension at the joint. It is to be noted from a comparison of Figures 6 and 7 that the fibres of the fan-shaped ligament, unlike those of the collateral ligament, are relaxed in flexion and progressively tighten as extension occurs. Attempted extension of the locked joint would therefore serve only to tighten the stranglehold and, if forcible, would result in rupture of the ligament or fracture of the osteophyte.

When oblique radiographs were taken of the metacarpal heads of our five patients they revealed the presence of hook-shaped osteophytes on the palmar surfaces of the metacarpal heads in all cases (Figs. 1 to 4).

We believe that the mechanism described above caused the locking in our patients. In Case 1 the dissector thrust into the joint acted in the manner of a shoe-horn and allowed the free edge of the ligament to negotiate the small osteophyte. In the other cases division of the palmar metacarpo-phalangeal ligament opened the encircling band. In Cases 4 and 5 the " cuff " was divided to one side of the palmar plate to reveal the offending osteophyte.

In the previously recorded cases the collateral ligament has most commonly been held responsible. These authors (Langenskiöld, Alldred, Aston) have, however, followed Poirier (1889) in using this term to include the " fan-shaped " fibres as well as the collateral ligament proper. Langenskiöld's diagrams specifically incriminate the former structure in his two patients. Alldred described a capsular tear along the palmar margin of the true collateral

ligament but his explanation for the locking is mechanically unsound, and we believe that his findings are better explained if the tear is considered as secondary to locking due to the mechanism we describe. Attempts by the patient or by the surgeon to extend a joint so locked would tend to tear the " fan-shaped " ligament from the collateral ligament at the site he describes. In one patient (Langenskiöld 1949) manipulation produced a fracture of the head of the metacarpal. The mechanism in Flatt's patient was very similar to the one we describe and differed only in the part played by a sesamoid bone in the palmar plate, which was caught in a bony ridge on the palmar surface of the head of the metacarpal. Aston suggested that in his patients the ulnar collateral ligament had been caught behind an osteophyte on the metacarpal head located at X in Figure 7. This explanation is mechanically sound, but in one of the two patients on whom he operated no such osteophyte is visible on the radiograph. The successful release of the finger in two of his patients resulting from division of the collateral ligaments can equally well be explained as the result of the simultaneous division of the " fan-shaped " ligaments, for he considers these structures as one.

TREATMENT

Manipulation of the locked joint if gentle is unlikely to be effective and if forceful may further damage the joint. We believe the joint should be explored from the palmar surface for it is there that the fault commonly lies. An oblique radiograph may confirm the presence of a palmar osteophyte. A small longitudinal incision through the transversely disposed fibres of the palmar metacarpo-phalangeal ligament where it forms the floor of the fibrous tendon sheath will cut the encircling band and free the joint without jeopardising its stability. As was demonstrated in Cases 4 and 5, the joint may be freed equally successfully by dividing the fan-shaped fibres at the lateral margin of the plate, thus obviating the necessity of opening the flexor tendon sheath.

SUMMARY

1. Five cases of locked metacarpo-phalangeal joint are described.
2. The anatomy of this joint is described and its bearing on the mechanism of locking discussed.
3. A method of treatment is suggested.

We are grateful to Mr J. C. Scott, Director of the Accident Service, Radcliffe Infirmary, Oxford, and to Mr J. D. Morgan of the Nuffield Orthopaedic Centre for permission to publish these cases.

REFERENCES

ALLDRED, A. (1954): A Locked Index Finger. *Journal of Bone and Joint Surgery*, **36–B,** 102.

ASTON, J. N. (1960): Locked Middle Finger. *Journal of Bone and Joint Surgery*, **42–B,** 75.

FLATT, A. E. (1958): Recurrent Locking of an Index Finger. *Journal of Bone and Joint Surgery*, **40–A,** 1,128.

GONTERMANN (1928): Durch Kapselinterposition fixierte Beugestellung im Zeigefingergrundgelenk nach spontan reposierter seitlicher Subluxation. *Archiv für Klinische Chirurgie*, **150,** 124.

JOSEPH, J. (1951): The Sesamoid Bones of the Hand and the Time of Fusion of the Epiphyses of the Thumb. *Journal of Anatomy*, **85,** 230.

LANGENSKIÖLD, A. (1949): Habitual Locking of a Metacarpo-Phalangeal Joint by a Collateral Ligament, a Rare Cause of Trigger Finger. *Acta Chirurgica Scandinavica*, **99,** 73.

POIRIER, P. (1889): Le doigt à ressort. *Archives Générales de Médecine*, **164,** 143.

GANGLION OF THE LATERAL POPLITEAL NERVE

KENNETH CLARK, STOKE-ON-TRENT, ENGLAND

From the Royal Infirmary, Edinburgh, Scotland

Cysts containing mucoid material and involving the lateral popliteal nerve as it lies in relation to the head of the fibula have been described on at least twelve occasions. The exact nature of the lesion has not been established.

Sultan (1921) reported the case of a patient with partial paralysis of the left lateral popliteal nerve due to cystic change. Treatment was by incision and evacuation of the contents of the cyst. A further case of partial paralysis from the same condition was reported by Zaar (1926). In two cases reported by Wadstein (1931) the cystic lesion had produced complete paralysis in the distribution of the anterior tibial nerve. In his first case the ganglion recurred after curettage and had to be dissected from the nerve; the second patient was treated initially by dissection and excision, partial recovery of function being obtained. The two cases reported by Ellis (1936) illustrated the relation of prognosis after operation to the duration of symptoms. In both cases symptoms had been present for one month, a rapid recovery of full function being obtained after the cyst had been emptied. A history of injury was given in one instance. Ferguson (1937) was able to dissect a bilocular cyst six centimetres long from the lateral popliteal nerve. Rapid recovery of function was obtained in twelve days; before operation there had been only slight weakness and paraesthesiae. He postulated mucinous degeneration of the connective tissue elements in the nerve as in ganglion. That complete paralysis can be produced in a short time was illustrated by Warren's (1946) case of complete paralysis after a history of only six weeks. A large multilocular cyst was resected and a graft inserted. Histological examination showed the lesion to be composed of connective and myxomatous tissue containing a cyst with a partial lining of synovial membrane. He ascribed the condition to a ganglion involving the connective tissue elements of the nerve.

Brooks (1952) described thirteen cases of nerve compression due to ganglion. In three cases the lateral popliteal nerve was compressed by a ganglion arising from the superior tibio-fibular joint. He suggested that ganglia of nerve sheaths and simple ganglia were anatomical variants of the same entity and considered that Sultan's case (1921) and Zaar's case (1926) were probably schwannomata. He refuted the suggestion that injury can produce the condition but stressed the importance of duration of compression in assessing the prognosis for recovery of function. Tupman (1957) reported a boy aged eighteen with sensory disturbance of six months' and drop foot of one month's duration; after surgical decompression of a large cystic swelling of the nerve complete motor and sensory recovery was obtained.

Ganglion of the lateral popliteal nerve, though rare, has thus evoked considerable discussion. Most authors have considered the condition to be one of simple ganglion involving the nerve and producing symptoms by compression of nerve fibres, though Jenkins (1952) included a similar type of case in his series of three neurilemmomata. There has been some doubt whether the ganglion developed in the nerve itself, as was suggested by Ferguson (1937), or arose from the superior tibio-fibular joint (Brooks 1952). The treatment most often recommended has been dissection of the cyst from the nerve. Simple evacuation of the cyst contents has once been followed by recurrence (Wadstein 1931). There are no reports available on the late results of treatment.

The present series comprises three cases of ganglion of the lateral popliteal nerve which have been under observation for over four years. Because all three cases were treated by resection of the involved area of nerve it has been possible to make a careful histological study in an attempt to establish the etiology of the condition.

CASE REPORTS

Case 1—A male railway clerk aged forty-two was admitted in September 1950 with a twelve months' history of pain over the outer side of the right leg from knee to ankle. Three months after the onset of symptoms a tender swelling was observed over the head of the fibula; it gradually enlarged. On examination there was a tender firm swelling five centimetres long in the line of the lateral popliteal nerve extending distally from the head of the fibula, with complete paralysis of the tibialis anterior muscle and partial paralysis of the extensor hallucis longus and extensor digitorum longus muscles, the peronei being unaffected. Sensation was preserved. At operation in September 1950 six centimetres of the lateral popliteal nerve were excised and primary suture was performed. The nerve was found to be infiltrated by multiple cysts of various sizes. No connection with the superior tibio-fibular joint was seen. Histological examination confirmed the diagnosis of ganglion. The patient made good progress after operation, and two years later there was full power in the peroneal muscles, good power in extensor digitorum longus and slight return of function in the extensor hallucis longus. Five and a half years later, however, he reported with a four weeks' history of pain in the right leg associated with a tendency to drag his toes. A nodular swelling in the line of the lateral popliteal nerve extending for three centimetres distally from the head of the fibula suggested a recurrence of the original lesion. At operation (April 1958) no evidence of recurrence was found but there was considerable fibrosis around the nerve. Histological examination of the fibrous tissue excised showed no evidence of recurrence of the ganglia.

Case 2—A male brewery worker aged fifty-two was admitted in January 1952 with a two months' history of increasing pain radiating from the outer aspect of the left knee to the dorsum of the foot, numbness of the leg and foot and an increasing tendency to drag the left foot. Three weeks before the onset of symptoms he had struck the back of his knee on a metal tap. Examination revealed a tender swelling four centimetres long and one centimetre across involving the lateral popliteal nerve at the level of the neck of the fibula. There was complete reaction of degeneration in the muscles supplied by this nerve and complete loss of sensation in its cutaneous distribution.

At operation in January 1952 the lateral popliteal nerve was found to be infiltrated by multiple cysts. A five-centimetre length was resected and the gap was closed by primary suture. The diagnosis of ganglion was confirmed by histological examination. The patient's progress was good and when he was seen in February 1958 he had full recovery of sensibility, and motor recovery as follows: peronei, 5; tibialis anterior, 3; extensor digitorum longus, 4; extensor hallucis longus, 0.

Case 3—A miner aged forty-two was admitted in October 1953 with a seven months' history of increasingly severe pain and paraesthesiae radiating from the outer side of the right knee to the foot. Foot drop had been noticed over the past five months. The patient spent most of his working day in the kneeling position, his left knee being protected by a pad retained by straps, one of which passed across the region of the head of the fibula. Two months before the onset of symptoms he had sustained a fracture-dislocation of the opposite knee. On examination there was a tender swelling fifteen by one and a half centimetres in the line of the lateral popliteal nerve distal to the head of the fibula. There was weakness of dorsiflexion and eversion of the foot and analgesia to pinprick in the lateral popliteal distribution. At operation in October 1953 the lateral popliteal nerve was found to be distended by multiple tense cysts over a distance of fourteen centimetres from the head of the fibula (Fig. 1). There was no connection with the superior tibio-fibular joint. The affected length of the nerve was resected, no repair being attempted. The diagnosis of ganglionic degeneration was confirmed histologically though a detailed serial section examination was not made because the specimen was preserved for exhibition. Follow-up examination in February 1958 revealed no evidence of recurrence.

TABLE I

FINDINGS IN RECORDED CASES OF GANGLION OF THE LATERAL POPLITEAL NERVE

Author	Sex	Age (*years*)	History of injury	Duration of symptoms (*months*)	Severity of paralysis	Operative findings
Sultan (1920)	M	56	Nil	Few	Partial	3 centimetres of cystic change in nerve
Zaar (1926)	M	22	Nil	2	Complete (motor and sensory)	6×2 centimetre cyst in the perineurium
Wadstein (1931)	M	36	Direct injury	8	Complete	Multiple cysts from gluteal fold to neck of fibula
	M	48	Nil	1	Complete	Cyst of size of little finger
Ellis (1936)	M	13	Nil	1	Complete	Multiple cysts
	M	44	Recurrent at work	1	Complete motor, incomplete sensory	Single cyst
Ferguson (1937)	M	15	Nil	½	Incomplete	6 centimetre trilocular cyst
Warren (1946)	M	29	Nil	1½	Complete	6 centimetre multilocular cyst attached to superior tibio-fibular joint
Brooks (1952)	M	53	Nil	1	Partial (motor and sensory)	Unilocular cyst with attachment to superior tibio-fibular joint
	F	67	Nil	48	Complete	Single cyst with attachment to superior tibio-fibular joint
	M	44	Nil	72	Partial motor, complete sensory	10 centimetres of cystic change
Tupman (1957)	F	18	Nil	6	Complete	Single large cyst
Present series	M	52	Direct injury	2	Complete	4 centimetre multiple cysts
	M	42	Recurrent at work	7	Partial (motor and sensory)	14 centimetre multiple cysts
	M	42	Nil	12	Partial motor only	6 centimetre multiple cysts

HISTOLOGICAL STUDIES

A serial section study has been made of two of the ganglia, the sections being stained by Mallory's trichrome stain and by haematoxylin and eosin.

Case 1—The cyst wall is composed of fibrous tissue with considerable degeneration of the intercellular substance which is greater towards the inner aspect of the cyst wall (Fig. 2). Two nerve bundles seen in the cyst wall can be traced throughout the full length of the specimen. An apparent lining membrane of the cyst (Fig. 3) is made up of degenerate fibroblasts. There is a noticeable increase in the number of nuclei towards the centre of the cyst, probably from loss of degenerate cytoplasm into the cavity of the cyst.

Case 2—The wall of the cyst contains much collagen. Towards the inner lining of the cyst the fibroblasts swell, their cytoplasm becomes basophilic and their nuclei undergo pyknosis and fragmentation. The cyst content is a homogeneous faintly basophilic material which has the appearance of mucin though it does not take up mucicarmine.

The histological picture described in these cases is like that of simple ganglion arising in relation to tendon sheaths and joint capsules. Nuclear proliferation or accumulation was described by Ogilvie (1948) as typical of simple ganglion.

TABLE I—*continued*

FINDINGS IN RECORDED CASES OF GANGLION OF THE LATERAL POPLITEAL NERVE

Histology	Treatment	Duration of follow-up (*months*)	Result
—	Incision and evacuation of contents	—	—
Cyst without endothelial lining	Dissection excision	—	—
Connective tissue rich in cells	Dissection excision	36	No motor recovery. Complete sensory recovery
Connective tissue like synovial tissue	Dissection excision	12	Complete recovery
—	Incision	11	Complete recovery
Fibrous tissue lining of cyst	Incision	3	Complete recovery
Mucoid degeneration as in ganglion	Dissection excision	6	Complete recovery
Myxomatous degeneration of fibrous tissue. Partial lining of synovial membrane	Dissection excision	—	—
—	Dissection excision	6	Complete recovery
Ganglion	Dissection excision	3	Incomplete sensory recovery. No motor recovery
Ganglion	Dissection excision	12	Complete sensory. Incomplete motor
Ganglion	Incision and evacuation	6	Complete recovery
Ganglion	Resection and primary suture	73	Complete sensory recovery. Incomplete motor recovery
Ganglion	Resection without repair	52	—
Ganglion	Resection and primary suture	91	Fibrosis requiring neurolysis after 8 years

DISCUSSION

The findings in the recorded cases of ganglion of the lateral popliteal nerve are summarised in Table I, which is as complete as the published data allow.

The nature of the lesion—The condition is generally thought not to be a new growth, though Jenkins (1952) reported one case which he considered to be the result of cystic degeneration in a neurilemmoma. A histological study of the present cases has failed to reveal any evidence of the nuclear arrangement seen in that tumour, such as the palisading of Antoni type A tissue or the haphazard reticulation of Antoni type B tissue in which cystic degeneration may occur (Antoni 1920). The present study suggests that the cystic change is in the nature of a ganglion arising in the supportive or connective tissue of the nerve. The histological characteristics of fibroblastic proliferation with progressive cellular degeneration and mucoid accumulation are those seen in the more common type of ganglion which arises from a joint capsule or tendon sheath though no communication may be traceable between the cavity of the cyst and that of any adjacent tendon sheath or joint (Ogilvie 1948). No connection with the tibio-fibular joint (Brooks 1952) has been noted in the present series, a careful histological study revealing ganglionic degeneration of the supporting tissues within the nerve itself. There

is little doubt that the interruption of nerve conduction is the result of compression of nerve fibres by the cyst formation, because the prognosis after excision or decompression is closely related to the duration of symptoms (Table I). Motor paralysis of more than two months' duration diminishes the possibility of full motor recovery.

The cause of the lesion—This remains in doubt, though a history of injury has been recorded in 25 per cent of the cases reported in the literature. A definite history of injury was given

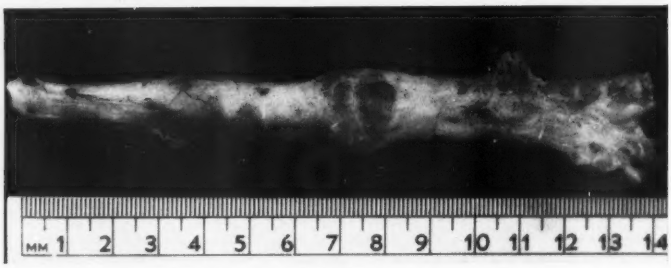


FIG. 1

Case 3—The length of nerve resected. Over twelve centimetres the nerve is distended by multiple cystic swellings lying within its substance.

FIG. 2

FIG. 3

Case 1. Figure 2—Transverse section of the cystic lesion, showing the fibrous nature of the cyst wall with its lining membrane. (×40.) Figure 3—Magnified view of lower left hand part of previous section, showing the nerve bundles lying in the cyst wall. Marked fibroblastic degeneration is obvious in two foci towards the inner lining, which is composed of degenerate fibroblasts. (×100.)

in one of the three cases reported in the present series; chronic pressure by straps worn at work was noted in another. There seems little doubt that the lateral popliteal nerve in its subcutaneous position adjacent to bone is often exposed to compression and irritation.

Treatment—This has varied considerably, as can be seen from Table I. Resection of the nerve was the procedure adopted in the cases here reported, because it was not thought possible to dissect the multiple cysts from the nerve fibres. As long as there is no evidence that the condition is a new growth the treatment should be that of dissection and enucleation when possible, and simple incision and expression of contents if dissection seems hazardous. Recurrence is possible after incision and expression (Wadstein 1931), but the recurrent cyst may be less extensive than the primary and thus amenable to dissection and enucleation.

SUMMARY

1. Three cases of ganglion of the lateral popliteal nerve are reported, all of which were treated by resection of the nerve.
2. In none was a connection between the ganglion and the superior tibio-fibular joint seen.
3. A careful histological study suggests that the condition is one of simple ganglion arising in the supporting tissues of the nerve.
4. A search of the literature has revealed twelve reported cases. The clinical and operative findings, together with the results of treatment, have been reviewed.
5. The treatment of choice is dissection of the ganglion from the nerve. If this proves difficult because of the multiplicity of cysts, incision and evacuation of cyst contents should be performed, although recurrence is possible after this procedure.
6. The prognosis for recovery of function is good when paralysis has been present for less than one year. Recovery of motor function is unlikely to be complete if there has been a longer duration of paralysis, though sensory recovery is usually good.

I wish to thank Sir Walter Mercer for permission to publish the cases reported in this paper. I am also indebted to Dr E. Isaacson, North Staffordshire Royal Infirmary, for his helpful criticism.

REFERENCES

ANTONI, N. R. E. (1920): *Ueber Rückenmarkstumoren und Neurofibrome.* München: J. F. Bergmann.
BROOKS, D. M. (1952): Nerve Compression by Simple Ganglia. *Journal of Bone and Joint Surgery*, **34–B,** 391.
ELLIS, V. H. (1936): Two Cases of Ganglia in the Sheath of the Peroneal Nerve. *British Journal of Surgery*, **24,** 141.
FERGUSON, L. K. (1937): Ganglion of the Peroneal Nerve. *Annals of Surgery*, **106,** 313.
JENKINS, S. A. (1952): Solitary Tumours of Peripheral Nerve Trunks. *Journal of Bone and Joint Surgery*, **34–B,** 401.
OGILVIE, R. F. (1948): *Pathological Histology.* Third edition, p. 426. Edinburgh: E. and S. Livingstone Ltd.
SULTAN, C. (1921): Ganglion der Nervenscheide des Nervus peroneus. *Zentralblatt für Chirurgie*, **48,** 963.
TUPMAN, G. S. (1957): Axonotmesis of Anterior Tibial Branch of Lateral Popliteal Nerve Due to Ganglion of the Nerve-sheath. *British Journal of Surgery*, **45,** 23.
WADSTEIN, T. (1931): Two Cases of Ganglia in the Sheath of the Peroneal Nerve. *Acta Orthopaedica Scandinavica*, **2,** 221.
WARREN, R. (1946): Ganglion of the Common Peroneal Nerve. *Annals of Surgery*, **124,** 152.
ZAAR (1926): Über Ganglien in der Nervenscheide. *Zentralblatt für Chirurgie*, **53,** 2,551.

INTRANEURAL GANGLION OF THE LATERAL POPLITEAL NERVE

ATHOL PARKES, GLASGOW, SCOTLAND

Invasion of the lateral popliteal nerve by a ganglion arising from the superior tibio-fibular joint seems to be by no means a rare occurrence, for no fewer than eight cases were seen and treated by the author within a period of just over six years.

CASE REPORTS

Of the eight patients described, seven were male and one female; their ages ranged from fifteen to seventy-four, with an average age of fifty.

Case 1—A milk delivery boy aged fifteen came to the out-patient department in January 1952 complaining of sudden onset of pain in the left knee. There was no history of injury and the pain was throbbing in character and worse at night. The only positive finding on examination was a tender swelling behind the head of the fibula and a provisional diagnosis of early osteomyelitis was made. The patient was kept under observation; after one week the pain had subsided and the patient was discharged.

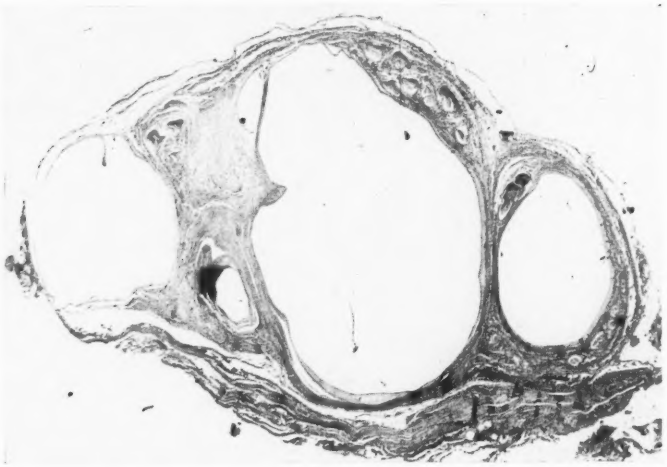


FIG. 1

Transverse section of lateral popliteal nerve containing multilocular ganglion. ($\times$ 5.)

In April 1952 he reported back with recurrence of the pain, which now radiated down the front of the leg to the dorsum of the foot. It was then realised that the tender swelling behind the head of the fibula was connected with the lateral popliteal nerve and the patient was referred to the author. Examination revealed that there was virtual paralysis of all the muscles of the anterior tibial compartment of the leg and disturbance of sensation in the distribution of the lateral popliteal nerve. A diagnosis of tumour of the lateral popliteal nerve was made.

At exploration in April 1952 the last inch of the lateral popliteal nerve was found to contain a quantity of yellowish jelly-like material which was within the sheath of the nerve and amongst the nerve bundles. The material extended for some distance into the two main

branches of the nerve. The pathological material was removed as completely as possible without sacrificing nerve fibres and the specimen was sent for biopsy. The report on the histological examination was as follows: " At places the tissue resembles the softer component of a Schwannoma-Ant. B and at other parts it could be taken for a fibro-lipoma. No definite Schwann cells could be made out."

Within a month of this operation the patient had regained full power in the anterior tibial muscles, but by April 1953 the swelling had recurred and persistent severe pain in the distribution of the lateral popliteal nerve necessitated further treatment.

In May 1953 the nerve was re-explored, when it was found that within the nerve sheath was a large multilocular cyst containing gelatinous fluid which now extended up to the middle of the popliteal space and down both branches of the nerve for several inches below the head of the fibula. Because of the recurrence and the severe pain it was decided to resect the whole of the affected portions of the nerves. The resulting permanent foot-drop was subsequently treated by transplantation of tibialis posterior to the front of the tarsus. The report on the histological examination of the nerves was as follows: " Multilocular cysts are present within the nerve sheath. The cysts are unlined or lined by cubical epithelium and contain mucoid material; they are situated between fascicles of nerve fibres. The appearances are those of a so-called ganglion of lateral popliteal nerve " (Fig. 1).

Case 2—A warehouseman aged forty-seven was referred from a medical neurological clinic in March 1954 with a six months' history of pain on the dorsum of the left foot and with weakness of dorsiflexion of the ankle of ten weeks' duration. He was found to have definite weakness of all the muscles of the anterior tibial compartment but full power in the peronei. There was diminution of sensation on the dorsum of the foot and a soft fleshy swelling was present on the lateral popliteal nerve behind the head of the fibula. A provisional diagnosis of Schwannoma was made.

At operation in March 1954 a typical ganglion was found within the nerve sheath. An attempt was made to remove it completely but during this procedure some of the nerve fibres were inevitably damaged. During the latter stages of removal it was found that the ganglion was connected by a small pedicle with the inferior aspect of the superior tibio-fibular joint. This pedicle was ablated.

Within six months of the operation the patient had regained full power in all the anterior tibial muscles. The patient was followed up for two years after operation and there was no sign of any recurrence.

Case 3—A forty-one-year-old commission agent was referred from another hospital where he had been under treatment for two years for prolapsed intervertebral disc. Treatment had included immobilisation in a plaster jacket, manipulation of the lumbar spine and other measures. His complaint of pain in the left knee radiating down the front of the leg to the dorsum of the foot had come on suddenly when he " jerked himself " in attempting to avoid colliding with a child in the street. About six months after the onset he had noticed weakness of dorsiflexion of the ankle and of extension of the toes. On examination a swelling was found on the lateral popliteal nerve behind the head of the fibula. Pressure on the swelling caused pain referred to the distribution of the nerve. There was complete paralysis of all the muscles supplied by the anterior tibial nerve but there was good voluntary power in the peronei. Sensation was diminished in the distribution of the lateral popliteal nerve. A diagnosis of ganglion of the lateral popliteal nerve was made and was confirmed at operation in October 1954. Again an attempt was made to dissect the ganglion from the nerve fibres and again the ganglion was found to have a pedicle half a centimetre in diameter connecting it with the inferior aspect of the superior tibio-fibular joint. The pedicle was divided and its stump cauterised with pure carbolic acid. After the operation there was complete motor and sensory paralysis of the lateral popliteal nerve. No recovery of function took place but

there was no palpable recurrence of swelling of the nerve during a follow-up of two years. The diagnosis of ganglion was confirmed by histological examination of the excised tissue.

Case 4—A retired man aged seventy-four was referred from a medical clinic in May 1955 with a nine weeks' history of pain in the right leg and foot and a seven weeks' history of weakness of dorsiflexion of the right ankle and of extension of the toes. The patient was found to have partial loss of function in the lateral popliteal nerve and there was a palpable swelling on the nerve in the region of the fibular head. A diagnosis of ganglion of the lateral popliteal nerve was made.

At operation in June 1955 a typical ganglion was found within the sheath of the nerve extending up from the level of the neck of the fibula for three inches. A pedicle was found connecting the ganglion with the superior tibio-fibular joint. This was divided and the contents of the ganglion were evacuated through multiple incisions in the nerve sheath. No attempt was made to excise the wall of the ganglion.

Eight months after the operation there was complete return of function in the nerve and four years afterwards there was no sign of any recurrence.

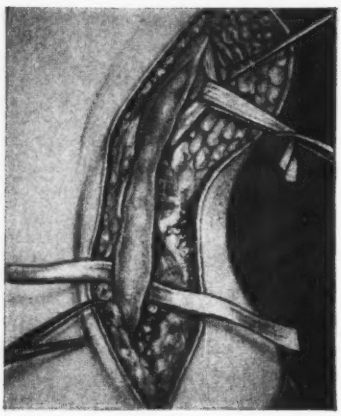


FIG. 2

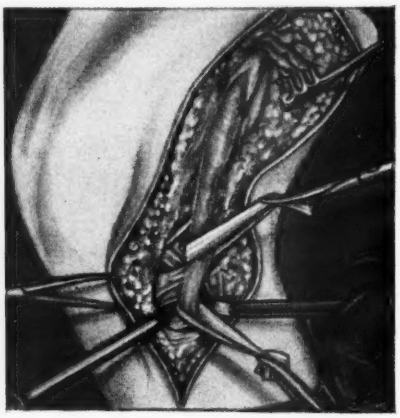


FIG. 3

Case 5—Figure 2—Invasion of the whole length of the lateral popliteal nerve by ganglion. The tape at the upper end of the field is round the medial popliteal nerve. Figure 3—The pedicle is seen joining the nerve between the two tapes.

Case 5—A sheet metal worker aged forty-seven was referred from another hospital in April 1956. He had complained of right-sided sciatica since early in 1953. In 1954 a myelograph had been done which had suggested the presence of arachnoiditis, but later that year a swelling was discovered on the lateral popliteal nerve in the region of the head of the fibula. In January 1955 the nerve had been explored by the late Mr Eric Paterson and was found to contain two cysts in its substance "each filled with clear gelatinous fluid such as is present in a ganglion." The cysts were evacuated completely. It was impossible to dissect out the cyst walls and it was thought that there was probably a narrow unidentified neck communicating with the joint. By November 1955 the muscles of the anterior compartment, which had been completely paralysed before the operation, were recovering, but in March 1956, following a strain, the swelling on the nerve enlarged and the anterior tibial muscles again became paralysed. There was hyperalgesia in the cutaneous distribution of the nerve.

In June 1956 the nerve was re-explored by the author when its whole length was found to contain a multilocular ganglion, the most bulbous portion of which was at its upper limit just below the origin of the lateral popliteal nerve from the sciatic (Fig. 2). A pedicle was found arising from the superior tibio-fibular joint (Fig. 3) and this was ligated and divided. No

attempt was made to excise the ganglion wall from the nerve, but the ganglion was decompressed by incising the nerve sheath at several levels.

Twenty-one months after this operation the patient had almost full power in all muscles and was free of pain. There was no recurrence of swelling of the nerve.

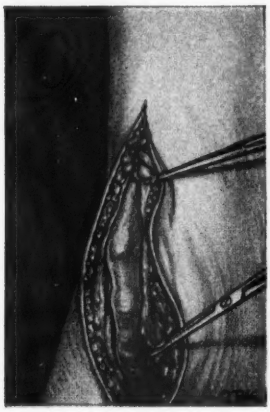


FIG. 4

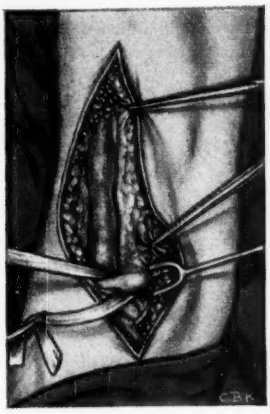


FIG. 5

Case 7—Figure 4—Multilocular ganglion in lateral popliteal nerve.
Figure 5—The tape is round the pedicle of the ganglion.

Cases 6, 7 and 8—These three patients were first seen between July 1956 and June 1958. All had clinical features similar to the above and all were treated by ablation of the pedicle, which was constant in position, and simple decompression of the ganglion by incision and expression of the contents. The appearance of the nerve in Case 7 is shown in Figures 4 and 5. All the patients recovered excellent function in the nerve and none had any recurrence of swelling on the nerve after at least fifteen months.

SUMMARY OF CLINICAL FEATURES

Pain—In nearly every case this was the initial symptom. Usually it was first localised to the region of the fibular head but later it was referred to the cutaneous distribution of the lateral popliteal nerve.

Paresis—Partial or complete paralysis of the muscles of the anterior compartment of the leg supplied by the anterior tibial nerve was always present. Seldom were the peroneal muscles affected.

Sensory disturbance—There was generally some blunting of sensation throughout the cutaneous distribution of the lateral popliteal nerve, but in one or two cases there was some hyperalgesia.

Palpable swelling of the lateral popliteal nerve behind the head of the fibula and sometimes extending well up into the popliteal space was present.

SUMMARY OF OPERATIVE PROCEDURES AND RESULTS

In Case 1 the presence of a pedicle was not suspected and it was not seen. The nerve was probably sacrificed unnecessarily.

After two operations at which the pedicle was not dealt with (Cases 1 and 5) the swelling recurred.

In no case was there recurrence of the swelling after the pedicle had been extirpated (seven cases) even when no attempt was made to remove the ganglion.

Satisfactory recovery of nerve function occurred in all except two cases. In one of these (Case 1) the nerve was excised, and in the other (Case 3) an attempt was made to excise the ganglion completely.

DISCUSSION

Pathology—This condition has been described before, notably by Brooks (1952), and it seems likely that some reported cases of mucoid degeneration of the lateral popliteal nerve have actually been intraneural ganglia. In attempting to understand the pathology of the condition there are two obvious difficulties: how does a ganglion arising from the superior tibio-fibular joint come to lie within the sheath of the lateral popliteal nerve, and why does the growth of such a ganglion seem to depend upon its connection with the joint?

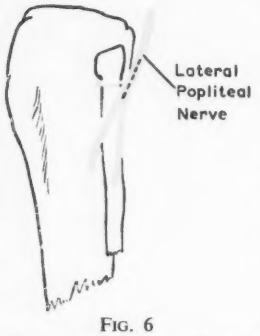


FIG. 6
Diagram showing articular branch supplying superior tibio-fibular joint.

The only possible answer to the first question seems to be that the ganglion initially tracks along the sheath of the small recurrent articular branch which is given off from the lateral popliteal nerve to the superior tibio-fibular joint and thus comes to lie within the sheath of the main nerve where it is free to enlarge and extend mainly in a proximal direction. The author has examined this articular branch in several cadavera and it certainly seems to correspond in position and direction with the pedicle of the ganglion as seen at operation (Fig. 6). The fact that the recurrent articular branch arises from that part of the trunk which is about to become the anterior tibial nerve probably accounts for the muscles of the anterior compartment being more severely affected than those in the lateral compartment.

The histology of one of the pedicles removed at operation (Case 7) was reported on as follows: " This is a thin-walled cyst containing clear sticky fluid. Microscopically the wall consists of dense acellular collagen with a simple synovial lining. At one part of the wall a small artery vein and nerve bundle is present " (Fig. 7).

It is of interest that the author has operated on two cases in which a dissecting ganglion was found within the adventitia of an artery. One was burrowing proximally within the sheath of the ulnar vessels in the lower forearm (Fig. 8) and had a pedicle which connected it with a carpal joint; the other was within the sheath of the radial artery on the dorsal aspect of the carpus. Its pedicle was emerging from between the bases of the first and second metacarpals and could easily have been arising from one of these carpo-metacarpal joints.

The other interesting question could, it seems, only be answered satisfactorily if one accepted the possibility of the gelatinous content of the ganglion originating in the joint and passing via the pedicle into the sac rather than being secreted by the wall of the sac itself. If this be so, then ganglia occurring elsewhere may perhaps not require complete excision of the sac provided that a pedicle can be located and removed.

Differential diagnosis—The possibilities of error in diagnosis are exemplified by the widely diverse provisional diagnoses with which the above patients were referred. These included osteomyelitis of the head of the fibula, prolapsed intervertebral disc, poliomyelitis, arachnoiditis, peripheral neuritis and Schwannoma. Provided a palpable swelling is sought and found on the lateral popliteal nerve, only the last-mentioned should cause any difficulty. In that case the diagnosis will become clear at exploration.

Treatment—It seems from this series that the correct treatment is early exploration, extirpation of the pedicle connecting the ganglion with the superior tibio-fibular joint and simple decompression of the multilocular cyst. Any attempt to remove the wall of the sac from within the nerve sheath is unnecessary and liable to damage nerve fibres.

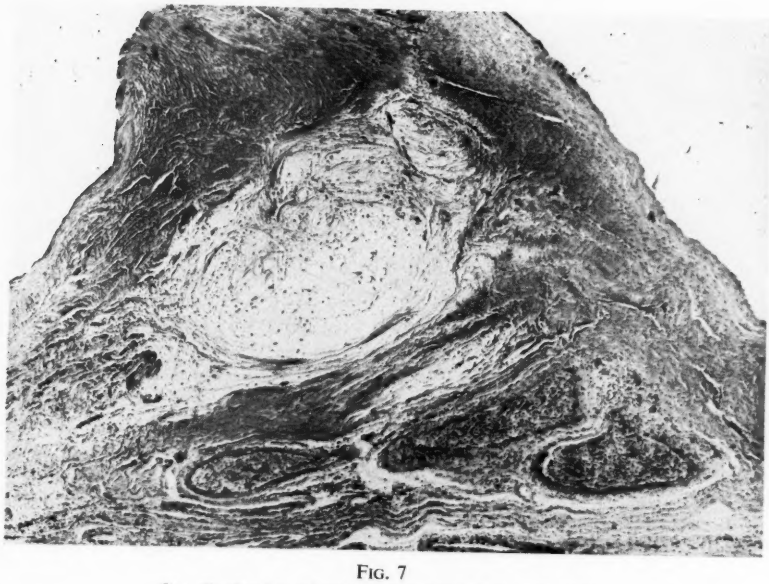


FIG. 7
Case 7—Section of wall of ganglion pedicle. (× 32.)

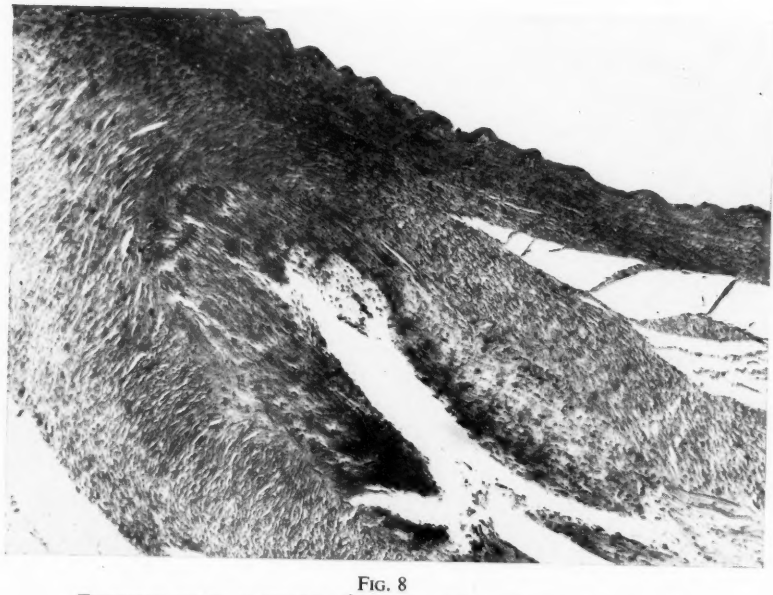


FIG. 8
Transverse section of ganglion within adventitia of ulnar vessels. (× 51.)

SUMMARY

1. Eight cases of intraneural ganglion of the lateral popliteal nerve seen at operation within a period of six years are described.
2. In seven of these a pedicle was found arising from the superior tibio-fibular joint—it was probably present in the remaining case.
3. The essential step at operation is to find and extirpate the pedicle.
4. The implications of these findings are discussed.

I wish to express my thanks to Dr Mary Catto for the histological examinations and for her help with the photomicrographs.

REFERENCE

BROOKS, D. M. (1952): Nerve Compression by Simple Ganglia. *Journal of Bone and Joint Surgery*, **34–B,** 391.

A METHOD OF ESTIMATING QUALITATIVE CHANGES OF BLOOD-FLOW IN BONE

A. McPherson,* John T. Scales† and L. H. Gordon,‡ London, England

From the Institute of Orthopaedics, Royal National Orthopaedic Hospital, London

The importance of obtaining information about the blood-flow in bone, its control and changes in disease, is evident. Many attempts to estimate bone blood-flow have been made and none has proved satisfactory. The disadvantages of using radioactive tracer elements to estimate blood-flow in bone have been well summarised by Boyd, Zilversmit and Calandruccio (1955) who pointed out that the amount of P^{32} in the bone depends on such factors as 1) the amount of P^{32} in the blood, 2) the amount of diffusion of P^{32} into the interstitial fluid, 3) the amount of exchange of interstitial P^{32} on the surface of the bone crystals, and 4) the amount incorporated into bone by metabolism. They concluded that the concentration of P^{32} in bone " is not strictly a measure of circulation." Edholm, Howarth and McMichael (1945) attempted to measure blood-flow in the humerus by venous occlusion plethysmography. This method, of necessity, ignores the rich supply of vessels other than the main nutrient arteries which contribute to the circulation in the humerus. One of the most promising attempts to estimate bone blood-flow was made by Braunsteiner and Grabner (1958), who used a heated thermocouple similar to that employed by Gibbs (1933), Grayson (1952) and Hensel and Ruef (1954).

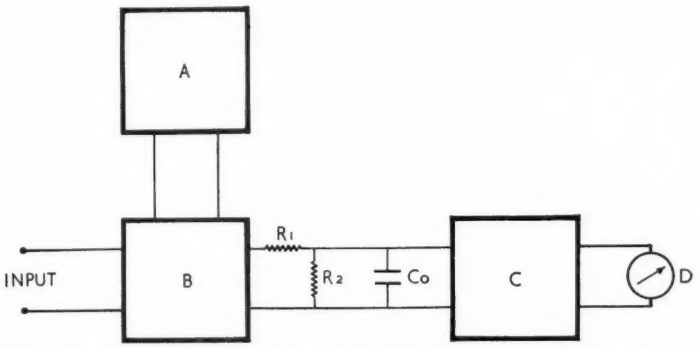


FIG. 1

Scheme of recording technique. Input from thermocouple probe. A—galvanometer modulator (Pye). B—amplifier and microvoltmeter (Pye). R_1—500 Ω R_2—150 k. Co—160 μ F. C—D.C. amplifier (Siemens-Ediswan). D—D.C. pen-recorder (Siemens-Ediswan).

The principle of the heated thermocouple is to use two thermocouples in opposition and in close proximity so that changes in the temperature of the tissue into which they are inserted do not change the resulting electromagnetic force. One of the thermocouples is then heated with a constant current so that its temperature is slightly above that of the tissue. If the blood-flow through the tissue increases, more heat is carried away from the heated thermocouple and the resultant changes in electromagnetic force or current can be measured. Braunsteiner

* External Scientific Staff, Medical Research Council.

† Department of Biomechanics and Surgical Materials, Institute of Orthopaedics, Royal National Orthopaedic Hospital, Stanmore, Middlesex.

‡ This work was undertaken during the tenure of a Travelling Fellowship awarded by the National Foundation, United States of America.

and Grabner (1958), using this method, examined the changes in blood-flow in the sternal marrow in man after the administration of adrenaline and nor-adrenaline. Their results are discussed later.

The present series of experiments were designed to discover the uses and limitations of the heated thermocouple technique in measuring bone blood-flow and whether there is any correlation between the blood flow and the intramedullary pressure.

METHODS AND MATERIALS

Thermocouple probes with an outside diameter of 0·83 millimetre were made in a manner similar to Mowbray's modification (1959) of the Hensel and Ruef (1954) needles. The manufacturing technique is described elsewhere (Sterrett 1961). A current sufficient to raise it from 37 to 38·5 degrees Centigrade was passed through the heater of the distal thermocouple from a well stabilised 6-volt alternating current supply. The output (which is very small) from the distal thermocouple was fed into a Pye D.C. amplifier and microvoltmeter (A and B, Fig. 1) and from the latter to a Siemens-Ediswan D.C. amplifier (C) and pen-recorder (D) through suitable resistors (R_1 and R_2) and a capacitator (Co).* The microvoltmeter was mounted on a vibration-free surface. No attempt was made to make the blood-flow recording quantitative.

The marrow pressure and arterial blood-pressure were recorded with Statham pressure transducers, fed through carrier amplifiers to Siemens-Ediswan D.C. amplifiers and pen-recorders.

The experiments were performed on healthy, adult cats anaesthetised with chloralose (80 milligrams per kilogram). Better results were obtained when the animals were heparinised, but this was not essential. In all experiments a thermocouple probe was inserted into the lower metaphysis of the femur and in some cases another thermocouple probe was inserted into the overlying quadriceps muscle. When the marrow pressure was measured the pressure head was connected to a metal tap inserted into the lateral side of the same femur at the junction of its uppermost and middle thirds. Arterial blood-pressure was recorded either from the contralateral femoral artery or from a carotid artery.

All results are based on at least fifteen consistent observations.

RESULTS

Effects of occlusion of femoral vessels and exercise—It was noticed that squeezing the muscles overlying the lower end of the femur increased the bone blood-flow. Similarly, occlusion of the ipsilateral femoral vein (Fig. 2) caused a marked increase in the blood-flow and was in fact used subsequently as an indication of the efficient functioning of the thermocouple. Occlusion of the femoral artery decreased the blood-flow (Fig. 2), and even occlusion of the artery for two minutes was not followed by any noticeable hyperaemia after release of the occlusion. The decrease in femoral blood-flow was less than might have been anticipated (see below). Applying a venous occlusion cuff above the site of the thermocouple (for example, at mid-thigh level) increased the bone blood-flow whereas occlusion below the site of the thermocouple (for example, at mid-leg level) decreased it. Passive movement of the knee decreased the bone blood-flow, but the results were often uncertain because of the unavoidable disturbance of the thermocouple.

Effects of afferent nerve stimulation—Pinching the skin, applying ice to the skin for thirty seconds, and electrically stimulating the central or peripheral cut end of either the sciatic or the femoral nerve while preventing muscle contraction by the administration of curare had no effect on the blood-flow in the bone.

* Pye and Co. Ltd. are now manufacturing heated thermocouples and blood-flow meters as a combined unit.

Intravenous and intra-arterial administration of adrenaline hydrochloride—Clinical adrenaline hydrochloride (a mixture of adrenaline and nor-adrenaline) was given in doses of about 0·3 microgram per kilogram through a cannula in a forelimb vein, or in doses of 0·1 microgram per kilogram into the ipsilateral femoral artery. The effects of adrenaline on the bone blood-flow were the same whether given intravenously or intra-arterially. The duration of these

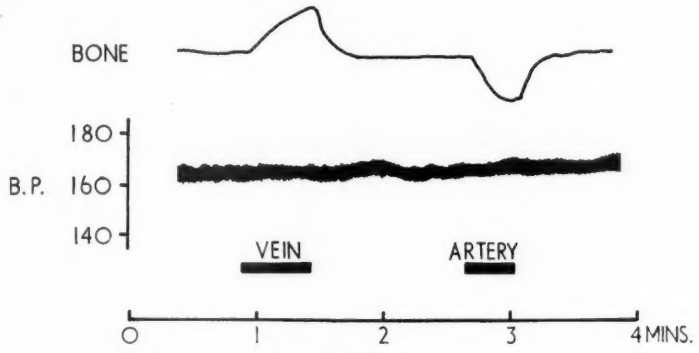


FIG. 2

Upper trace-recording of changes in the blood-flow in the lower end of the femur—increase shown upwards. Lower trace—arterial blood pressure (millimetres Hg.). At the first signal mark the femoral vein was temporarily occluded and at the second signal mark the femoral artery was temporarily occluded.

effects varied from animal to animal. Figures 3 and 10 illustrate a typical response in which 1 microgram of adrenaline produced a transient rise in arterial blood-pressure which was usually followed, within a few seconds, by a slight decrease in bone blood-flow and a slight increase in muscle blood-flow. This initial response was consistently followed by an increase in bone blood-flow which lasted for up to ten minutes and was accompanied by a decrease in muscle blood-flow which continued after the arterial blood-pressure had returned to its previous level.

In some experiments the cats were given 10 micrograms adrenaline intravenously so that the results could be compared with those of Braunsteiner and Grabner and with those of Stein, Morgan and Porras (1958), who used enormous doses when examining the marrow pressure in dogs. Figure 4 shows the results of such an experiment. The changes in bone blood-flow preceded the changes in arterial blood-pressure and consisted in a slight increase followed by a larger decrease in flow. These changes were followed by a further increase and then a prolonged decrease in flow.

Intravenous and intra-arterial administration of nor-adrenaline bitartrate—Nor-adrenaline bitartrate was given intravenously in doses of about 1–2 micrograms per kilogram and intra-arterially in doses of 0·3 microgram per kilogram. The effects on bone blood-flow were similar in both cases. There was little

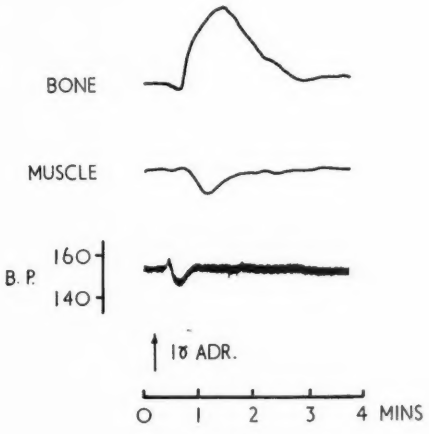


FIG. 3

Upper trace—the bone blood-flow (femur). Middle trace—the muscle blood-flow (quadriceps). Bottom trace—the arterial blood-pressure (millimetres Hg.). At the signal mark 1γ adrenaline hydrochloride was injected intravenously.

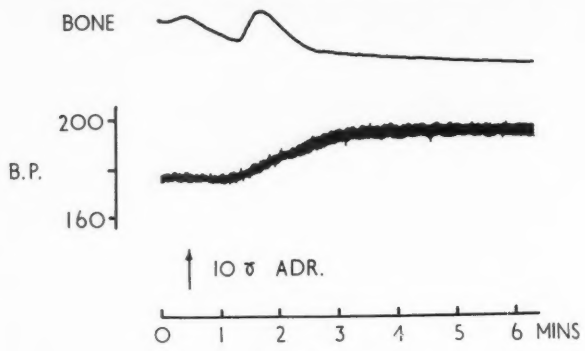


FIG. 4

Upper trace—the bone blood-flow (femur). Lower trace—the arterial blood-pressure (millimetres Hg.). At the signal mark 10 γ adrenaline hydrochloride was injected intravenously.

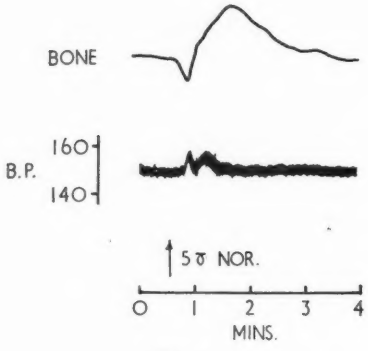


FIG. 5

Traces as in Figure 4. At the signal mark 5 γ nor-adrenaline bitartrate was injected intravenously.

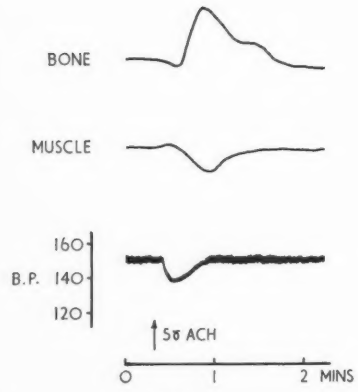


FIG. 6

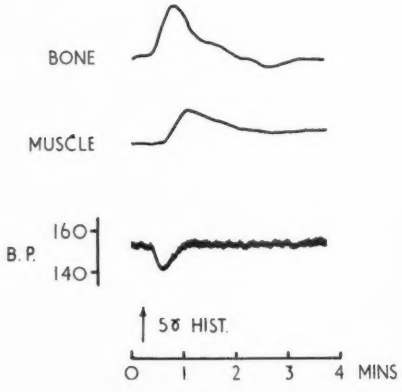


FIG. 7

Figure 6—Traces as in Figure 3. At the signal mark 5 γ acetylcholine chloride was injected intravenously. Figure 7—Traces as in Figure 3. At the signal mark 5 γ histamine acid phosphate was injected intravenously.

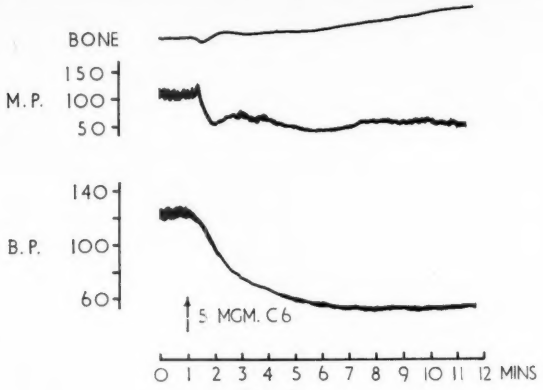


FIG. 8

Upper trace—the bone blood-flow (femur). Middle trace—the marrow pressure (millimetres Hg.). Lower trace—the arterial blood-pressure (millimetres Hg.). At the signal mark 5 milligrams hexamethonium bromide was injected intravenously.

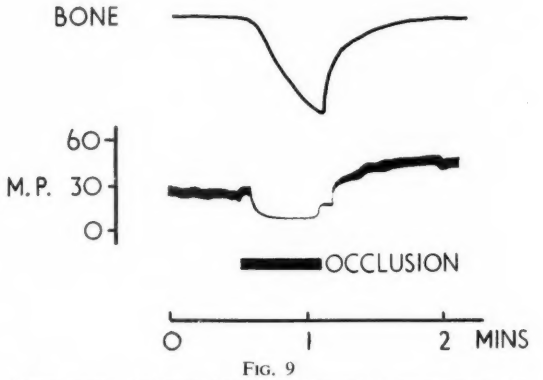


FIG. 9

Upper trace—the bone blood-flow (femur). Lower trace—the marrow pressure (millimetres Hg.). For the duration of the signal mark the femoral artery was occluded.

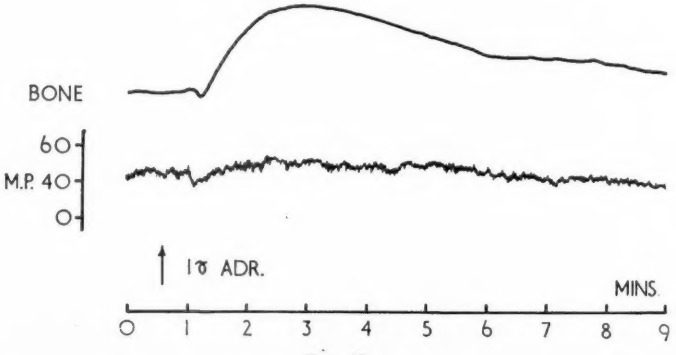


FIG. 10

Upper trace—the blood flow (femur). Middle trace—the marrow pressure (millimetres Hg.). At the signal mark 1 γ adrenaline hydrochloride was injected intravenously.

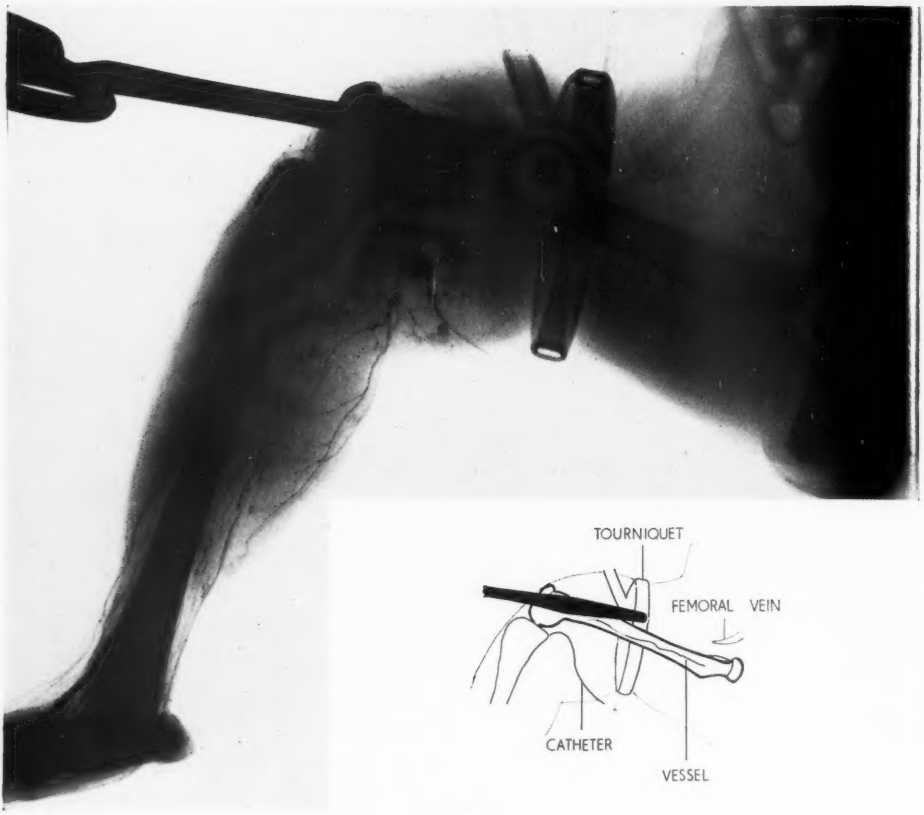


FIG. 11

Radiograph taken a few seconds after injection of 3 millilitres " Hypaque " through a catheter into the anterior tibial artery while a tight tourniquet was constricting the mid-thigh. No radio-opaque material is visible in the soft tissues immediately above the tourniquet, but a vessel is visible in the femur and the femoral vein is partly visible.

difference in the effects of nor-adrenaline and adrenaline on bone blood-flow except that nor-adrenaline did not increase the blood-flow so much as adrenaline, nor did the increase persist so long. Nor-adrenaline also usually produced a more marked initial decrease in flow (Fig. 5).

Intravenous administration of acetylcholine chloride—Acetylcholine was given only intravenously and in doses of 1·5 micrograms per kilogram. The increase in bone blood-flow produced by acetylcholine was less marked than that produced by adrenaline and was of considerably shorter duration (Fig. 6). It coincided with the onset of the changes in blood-flow in the overlying muscle and was of similar duration.

Intravenous administration of histamine acid phosphate—Histamine was given intravenously in doses of 1–2 micrograms per kilogram. It also caused an initial increase in bone blood-flow lasting for two to three minutes (Fig. 7), which was sometimes followed by a more prolonged decrease in flow. Curiously, the increase in blood-flow in bone usually preceded the increase in flow in the overlying muscle. The latter persisted for a much longer time than the increase in bone blood-flow.

Intravenous administration of hexamethonium bromide—Intravenous administration of hexamethonium bromide in doses of 5 milligrams produced a prolonged increase in

the bone blood-flow (Fig. 8) which persisted as long as the decrease in arterial blood-pressure.

The relationship of changes in bone blood-flow to changes in marrow pressure—Although bone blood-flow and marrow pressure were not investigated simultaneously in a large enough number of experiments, there did not seem to be any consistent correlation between them. In relatively simple procedures such as occluding the femoral artery the marrow pressure fell with the blood-flow (Fig. 9). When the femoral vein was occluded the two increased. However, it was possible to demonstrate quite considerable changes in bone blood-flow without any great changes in marrow pressure. In the experiment illustrated in Figure 10, 1 microgram

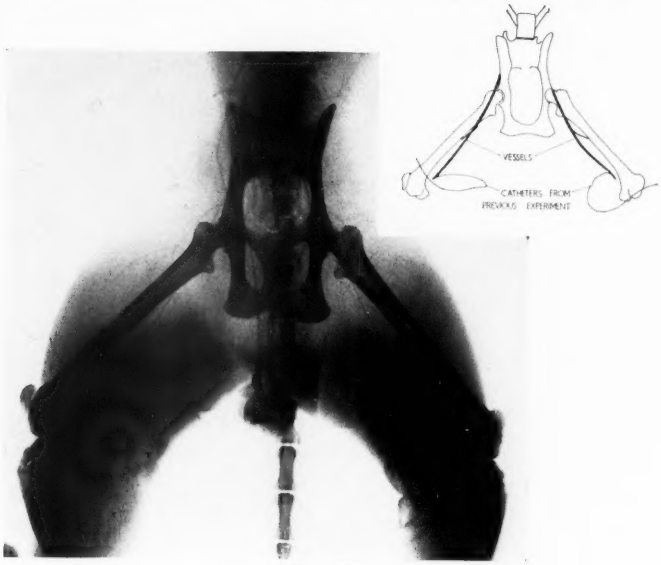


FIG. 12

Radiograph taken a few seconds after the injection of 5 millilitres " Hypaque " into a renal artery with the aorta tied off just above the bifurcation. The femoral and profunda femoris arteries are visible on both sides. (The two catheters visible at the lower end of the femur were used in a previous, unrelated experiment.)

adrenaline hydrochloride produced a considerable and prolonged increase in bone blood-flow although the marrow pressure increased only slightly.

Control experiments—In an attempt to assess the value of the measurement made by heated thermocouples, two other experiments were performed as a control. The increase in bone blood-flow that occurred when the venous return was obstructed proximally suggested that venous blood was " shunted " from the soft tissues through the only remaining outlet, the bone. An attempt was made to demonstrate this shunting radiographically. A fine polythene cannula was inserted into the anterior tibial artery of a cat and a tight tourniquet was applied at mid-thigh level. A small amount of " Hypaque " was then injected into the artery and radiographs were taken within a few seconds. Figure 11 illustrates the result of one of these experiments, in which no " Hypaque " appeared in the soft tissues for a few centimetres above the tourniquet but a vessel was visible in the bone. Sufficient " Hypaque " had returned by this route to partly fill the femoral vein. These experiments are interesting in that they were suggested by the blood-flow investigations and because Steinbach, Jergesen, Gilfillan and Petrakis (1957) stated: " All attempts to follow roentgenographically the arterial and venous circulation through bone after injection of a major afferent artery with opaque liquids have been unsuccessful."

A further experiment was suggested by the results of arterial occlusion. As previously stated, the decrease in bone blood-flow on occluding the femoral artery was not so great as might have been expected. Further experiments showed that clamping the aorta at its bifurcation produced a greater reduction in bone blood-flow, and an even greater reduction was caused by clamping it just below the renal arteries. Experiments were therefore performed in which cats were eviscerated and a cannula was inserted into a renal artery. The aorta was then tied off tightly below the ilio-lumbar arteries and immediately above the bifurcation. "Hypaque" was then injected into the renal artery and radiographs were taken of the lower limbs. Figure 12 illustrates the results of one of these experiments; the contrast medium has found its way through by-passes to the femoral artery and the profunda femoris.

Although these experiments do not check the accuracy or consistency of bone blood-flow measurement by heated thermocouples, they do suggest that the latter may provide useful information which it is possible to confirm by other means.

DISCUSSION

Limitations of the method—There are many limitations to the use of heated thermocouples to measure blood-flow. The thermocouple probe can sample only a limited amount of tissue and may reflect only a purely local change in blood-flow (for example, in an arteriole close to the end of the probe), or perhaps no more than a redistribution of blood in the tissue. Other limitations are the possibility of a clot forming around the probe and decreasing its sensitivity; its great sensitivity to movement of the animal or of the investigator; and the impossibility, at present, of making the method quantitative.

One particular difficulty is worth discussing. If the instrument is inserted in such a way that the distal thermocouple is pushed against the cortical bone, then this thermocouple records little change in blood-flow and the record is inverted, that is, the more proximal opposing thermocouple, which normally just "backs off" any local tissue temperature changes, becomes the recording thermocouple and an increase in blood-flow in its neighbourhood moves the pen-recorder in the opposite direction to that expected.

It is with these limitations in mind that the results must be considered.

Discussion of the results—The results of vascular obstruction, of passive exercise and nerve stimulation suggest that, to a considerable extent, the blood-flow in bone passively reflects the blood-flow changes in overlying muscle. Restricting the venous return above the level of the thermocouple increases the bone blood-flow. This suggests that blood is then shunted from muscle to bone. The passive nature of such changes in bone blood-flow is further indicated by the absence of any effect from nerve stimulation. However, the blood-flow in bone does not always follow the blood-flow in muscle. Thus, when small doses of adrenaline or acetylcholine were given intravenously the muscle blood-flow decreased and the bone blood-flow increased. By contrast, when histamine was administered the muscle blood-flow and the bone blood-flow both increased.

The bone blood-flow can change independently of the general arterial blood-pressure. In most of the experiments in which drugs were administered the changes in bone blood-flow persisted for minutes after the blood-pressure had returned to its previous level. This may mean that adrenaline, acetylcholine and histamine exert a local effect on blood vessels supplying the bone. It is significant that Stein *et al.* (1958) found that adrenaline, nor-adrenaline or pituitrin produced a local effect on marrow pressure independent of the systemic blood-pressure changes.

It is difficult to compare Braunsteiner and Grabner's investigations with our own because they worked on man and on the sternal marrow. They found that after intravenous doses of 10–50 micrograms of adrenaline the sternal blood-flow changed in two phases. During the first the blood-flow increased and then decreased, and during the second phase the blood-flow

slowly increased and remained so for a considerable time. The first phase described by Braunsteiner and Grabner is similar to that in the experiments illustrated in Figure 4, and the second phase is similar to that illustrated in Figure 9.

Allowing for differences in species and dosage and possible differences in the nor-adrenaline content of clinical adrenaline, these results are comparable. The results that Braunsteiner and Grabner described after injecting nor-adrenaline intravenously are indeed exactly the same as those found in the present experiments, that is, an initial decrease in bone blood-flow followed by an increase.

It is even more difficult to compare the changes in bone blood-flow produced by drugs and the changes in marrow pressure described by Stein *et al.* because the latter used drugs in such large doses. However, there does not appear to be a consistent relationship between bone blood-flow and marrow pressure.

The relationship between bone blood-flow and marrow pressure requires further investigation because, if it is possible to discover the relationship between the two, it would be possible to estimate the bone blood-flow by pressure measurements which are much more simple and convenient to perform than thermocouple investigations.

The demonstration of a patent vessel in the femur of the cat after injection of a radio-opaque material into the anterior tibial artery with a tight tourniquet applied to the thigh is worthy of further investigation because this might be of clinical significance. Similarly, the demonstration of an alternative vascular route to the lower limbs after aortic occlusion suggests the possibility of an arterial plexus similar to the vertebral venous plexus, and further investigation of this is being undertaken.

SUMMARY

1. A method of recording changes in bone blood-flow using a heated thermocouple is described.
2. Occlusion of the femoral artery or aorta decreases the blood-flow in the femoral metaphysis.
3. Occlusion of the femoral vein and intravenous injection of adrenaline, nor-adrenaline, acetylcholine, histamine or hexamethonium bromide increase the blood-flow in the femoral metaphysis.

We would like to thank Mr H. J. Seddon for his advice and encouragement, Dr J. F. Mowbray for his advice, Mr S. J. Sterrett of the National Institute for Medical Research for making the thermocouple probes, Mr G. Pierson and Mr D. Marlborough for their technical assistance, and Mr R. J. Whitley for the photographs.

REFERENCES

BOYD, H. B., ZILVERSMIT, D. B., and CALANDRUCCIO, R. A. (1955): The Use of Radio-active Phosphorus (P^{32}) to Determine the Viability of the Head of the Femur. *Journal of Bone and Joint Surgery*, **37–A,** 260.

BRAUNSTEINER, H., and GRABNER, G. (1958): Der Einfluss von Adrenalin und Noradrenalin auf die Durchblutung des Knochenmarks. *Zeitschrift für die gesamte experimentelle Medizin*, **130,** 289.

EDHOLM, O. G., HOWARTH, S., and MCMICHAEL, J. (1945): Heart Failure and Bone Blood Flow in Osteitis Deformans. *Clinical Science*, **5,** 249.

GIBBS, F. A. (1933): A Thermoelectric Blood Flow Recorder in the Form of a Needle. *Proceedings of the Society for Experimental Biology and Medicine*, **31,** 141.

GRAYSON, J. (1952): Internal Calorimetry in the Determination of Thermal Conductivity and Blood Flow. *Journal of Physiology*, **118,** 54.

HENSEL, H., and RUEF, J. (1954): Fortlaufende Registrierung der Muskeldurchblutung am Menschen mit einer Calorimetersonde. *Pflügers Archiv für die gesamte Physiologie*, **259,** 267.

MOWBRAY, J. F. (1959): Measurement of Tissue Blood Flow Using Small Heated Thermocouple Needles. *Journal of Applied Physiology*, **14,** 647.

STERRETT, S. J. (1961): Improved Manufacturing Technique for Blood-flow Probes. *Journal of Scientific Instruments*, **38,** 59.

STEIN, A. H., Jun., MORGAN, H. C., and PORRAS, R. F. (1958): The Effect of Pressor and Depressor Drugs on Intramedullary Bone-Marrow Pressure. *Journal of Bone and Joint Surgery*, **40–A,** 1,103.

STEINBACH, H. L., JERGESEN, F., GILFILLAN, R. S., and PETRAKIS, N. L. (1957): Osseous Phlebography. *Surgery, Gynecology and Obstetrics*, **104,** 215.

THE VASCULAR CONTRIBUTION TO OSTEOGENESIS

IV. The Effect of Pressure upon the Epiphysial Cartilage of the Rabbit

JOSEPH TRUETA, OXFORD, ENGLAND and ANTONI TRIAS,* CHICAGO, UNITED STATES OF AMERICA

From the Nuffield Orthopaedic Centre, Oxford

In the previous paper of this series (Trueta and Amato 1960) the effects of reducing or suppressing the blood flow to either the epiphysial or the metaphysial side of the growth cartilage were reported. The most striking findings were the extreme dependence of the life of the chondrocytes—the only known agents of longitudinal growth of long bones—on the blood supply of the epiphysis and the lack of participation of the metaphysial blood supply in maintaining the life of the cells of the epiphysial cartilage. It was found that, on the contrary, the vascular activity of the metaphysial side of the growth cartilage was responsible for the death and removal of hypertrophic cartilage cells, for when the blood flow of these metaphysial vessels was discontinued calcification was prevented and the hypertrophied chondrocyte survived for a long time.

When this finding was made, early in our research, it became interesting to investigate the part that pressure plays in the alteration of the normal rate of blood supply adjacent to the growth cartilage. It also was considered important to collect further data on the influence of pressure on the mechanism of growth apart from its action upon the vessels.

MATERIALS AND METHOD

For this work, as for the preceding one, the rabbit only was used. All the animals were from six to eight weeks old (except numbers 14 and 15 which were ten and twelve weeks respectively); their weights ranged from 1·25 to 1·5 kilograms. The rabbits were prepared by the technique described by Trias (1961). As in his work, compression of the epiphysial cartilage was produced by the use of a clamp with springs and pins aseptically placed above and below the growth cartilages of the right distal femoral and proximal tibial epiphyses (Fig. 1), the left hind limb being used as a control. To prevent flexion at the knee, and consequent decrease of pressure across the growth cartilage, a plaster reaching from foot to groin was applied in most experiments. In a few the knee was allowed to flex, so that the area of greatest pressure across the epiphysial cartilage shifted progressively backward. This displacement of the areas of greatest pressure permitted a study of the changes produced in the growth cartilage by the constricting force in varying positions. Radiographs of both hind limbs were taken at the end of the procedure.

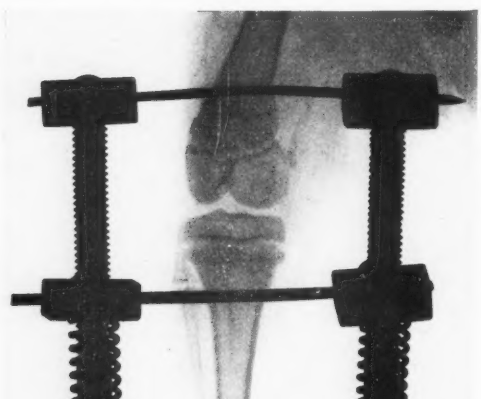


FIG. 1
Apparatus for compression of the knee.

* Formerly Girdlestone Scholar in Orthopaedic Surgery.

The animals were killed with an overdose of ether. This was followed by an intra-arterial injection of a mixture of equal parts of 2 per cent Berlin blue and Micropaque (barium suspension) or, in some animals, Indian ink. The amount varied according to the weight of the animal; on average some 40 millilitres of the mass were perfused.

After removal of the soft tissues and disarticulation of the tibia and femur of both limbs the four bones were radiographed again. Finally, the distal ends of the femora and the proximal ends of all the tibiae were embedded in celloidin, and histological sections were cut and prepared by the methods described by Trueta and Harrison (1953).

Of the thirty-one rabbits used in this experiment, two died from anaesthesia at operation and another was discarded because the position of the clamps was unsatisfactory.

Table I shows the number and duration of experiments and the survival time after removal of the pins and clamp.

TABLE I

DURATION OF COMPRESSION

Days . . .	3	4	5	7	8	9	10	12	13	14	15	17	20	21
Number of animals	1	1	2	5	2	1	1	3	2	2	1	2	1	2

Nine of these rabbits were allowed to survive for a varying length of time after the removal of the pins and clamp at different stages. The shortest survival time was two days and the longest seventy-five days. In describing our findings we will refer particularly to this group.

Measurement of pressure—The use of springs between the head of the pressure clamp and the pin nearest it causes a compressing force which we have attempted to measure. It also allows for some expansion of the area between the pins. This precaution was considered necessary because of the excessive increase in pressure which would be caused by the division and growth of the cells of the growth plate.

A force of between 3·5 and 4·5 kilograms was required to shorten our springs, unloaded, to half their length. Since each clamp contained two similar springs, the compressing force acting on the pins varied from 7 to 9 kilograms (15 to 20 pounds) when the springs were reduced to half their length. Since the pins were placed at an almost constant distance from the growth cartilages, the pressure upon these was estimated from the formula $P=\frac{F}{S}$, where P=pressure, F=force and S=surface area. The area of contact between the two opposing epiphyses was considered of the order of one-third of a square centimetre. Therefore $P=8\times\frac{3}{1}$ (average)=24 kilograms per square centimetre. Since the growth cartilages were situated much closer to the pins than was the area of pressure in the joint surfaces, a substantial increase in the area of distribution of pressure at the level of the growth cartilages had taken place (Fig. 2). This meant that the cone of pressure had increased to at least twice its size, thus making the formula $P=\frac{8}{1}$ centimetres=8 kilograms per square centimetre or somewhat less. This resulted in less damage to the growth cartilage than that caused to the articular

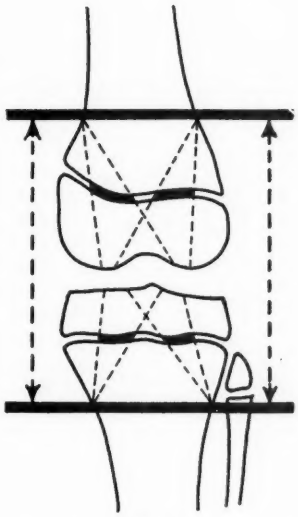


FIG. 2
Diagram showing the "cones of pressure."

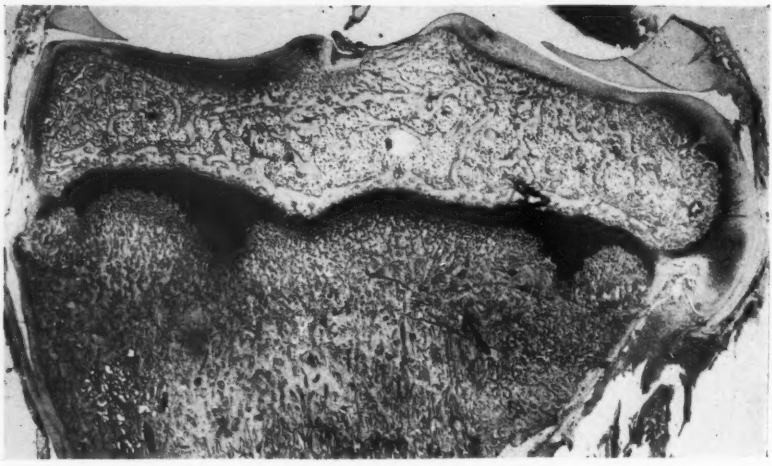


FIG. 3
Triangular thickening of the plate limited to the areas of greatest pressure. (×20.)

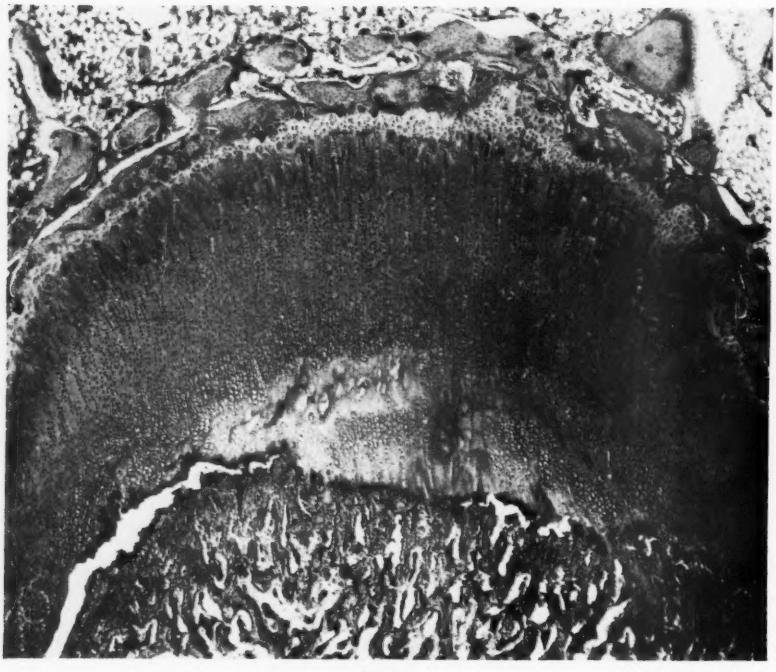


FIG. 4
Widened epiphysial femoral cartilage with a central area of degeneration. (×120.)

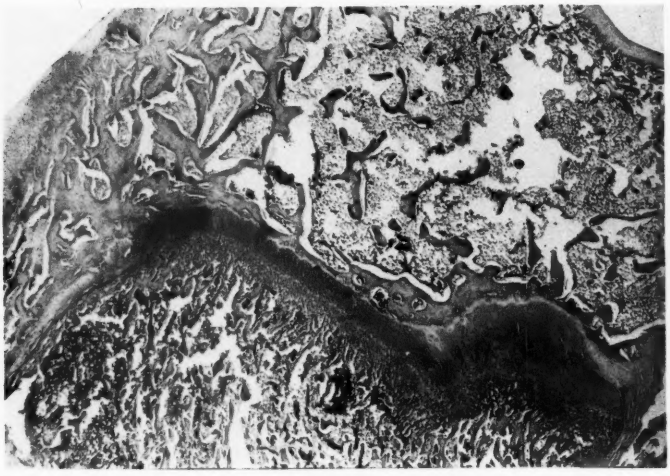


FIG. 5
Thinning of growth plate on the left while on the right the widening still persists. (×25.)

FIG. 6
Vascular bridge preceding the bone formation across the bone plates. (×240.)

cartilage by the same compressing force. Nevertheless, it maintained the greatest changes in that part of the growth cartilage which was intersected by the cone of pressure. In the study of these changes it was fortunate that in a number of animals the position of the tibia changed during the experiment, for it allowed us to study the changes of the epiphysial cartilage in every position, including extreme flexion.

RESULTS

After the application of persistent pressure of the nature described, the most common finding in the epiphysial cartilage was an increase in its height, frequently limited to the areas of greatest pressure. The earliest increase detected was found to occur after about three days and affected both the femoral and the tibial growth cartilages. At this early stage the increase affected only the restricted area of greatest compression—that intersected by the cone of pressure (Fig. 3). After seven days of pressure the whole area of the epiphysial cartilage became widened in almost all cases. In some rabbits signs of disorganisation of the columnar arrangement of the growth plate were observed in the areas of strongest pressure (Fig. 4); different segments of the columns began losing their boundaries and it became difficult to distinguish between the proliferative and the hypertrophic zones. The nuclei were not stained and finally the cell limits were no longer visible.

In animals with the clamps left for longer periods the whole of the compressed area of the epiphysial cartilage broke down and in these rabbits in which the pressure was maintained for durations up to a maximum of twenty-one days, the growth plate became thinner and thinner (Fig. 5), until it was seen invaded by vessels and eventually cut by a long bridge of bone between epiphysis and metaphysis (Fig. 6).

Mechanism of epiphysial cartilage thickening—It was of particular interest to investigate the way in which the growth cartilage increased in height. As mentioned above, this increase was the earliest change detectable in the growth cartilage under pressure. It was not due so much to the chondrocytes getting bigger but to their getting more numerous. Repeated counts in all the animals studied gave a constant histological picture of the part taken in the increase by the different sections of the epiphysial cartilage. No change was found in the number of germinal and proliferative cells remaining within the normal limits of twenty to thirty (Trueta and Morgan 1960). On the other hand, the number of hypertrophic cells varied in accordance with the increase in total thickness of the cartilage, amounting in some cases to as many as eighty or ninety (Fig. 7) instead of the normal ten to twelve. Beyond that number we could not find a further increase. It appeared that a point was reached beyond which the cells of the hypertrophic zone could not remain alive as they became squashed against each other and against a fibrous barrier which appeared at the distal end of the columns (Fig. 8) until the cells degenerated and finally died.

After the examination of some hundreds of sections it became apparent that those thin growth cartilages examined in the late stages of degeneration and disorganisation in all probability had been preceded by an earlier phase of cartilage thickening.

When the hypertrophic cartilage cells remained for a longer time without being removed the enormously enlarged columns often lost their ability to grow straight and became curved with their convexity towards the periphery of the metaphysial region (Fig. 9).

The metaphysial end of these enormous columns was covered by a band of degenerate fibrous tissue well supplied with vessels at its deepest side. As this fibrous band was only found where the hypertrophic cells were firmly compressed against each other, we believe the death of these cells to be due to their inability to carry on their required interchanges in such a confined area. Sometimes chondrocytes could still be detected in their fibrous band and even beyond (Fig. 10).

The examination of sections from areas of growth cartilage affected by severe disorganisation and vascular invasion showed that, without exception, the compression had

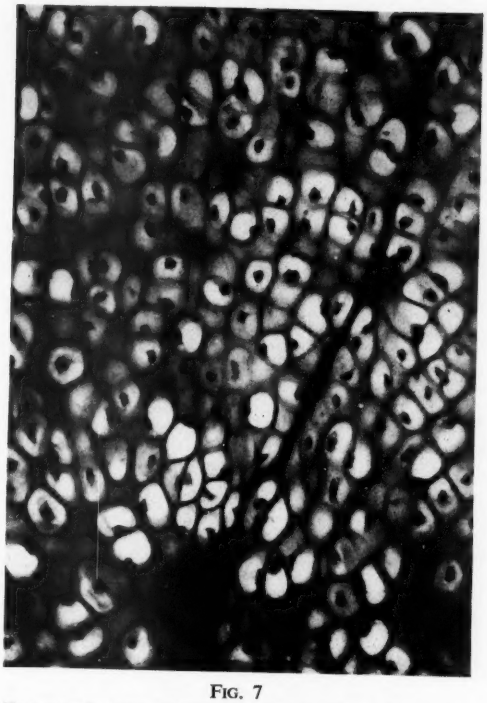


FIG. 7
Enormously long columns of hypertrophic cells caused by compression. (×300.)

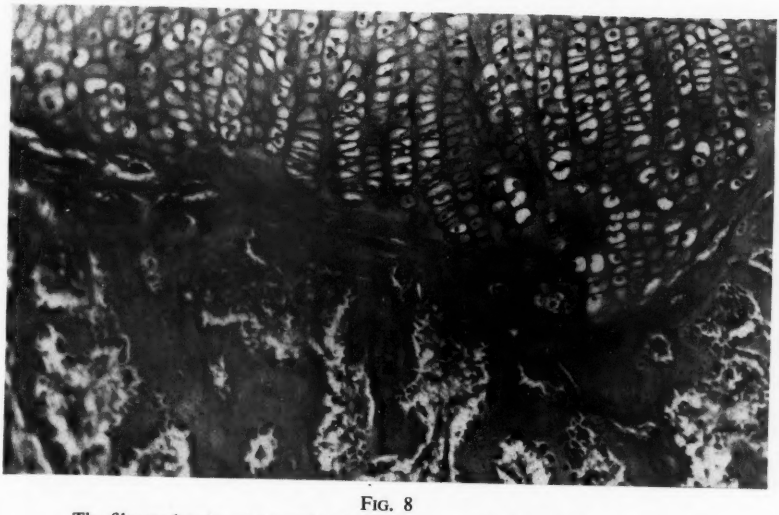


FIG. 8
The fibrous barrier at the ends of the columns of hypertrophic cells. (×90.)

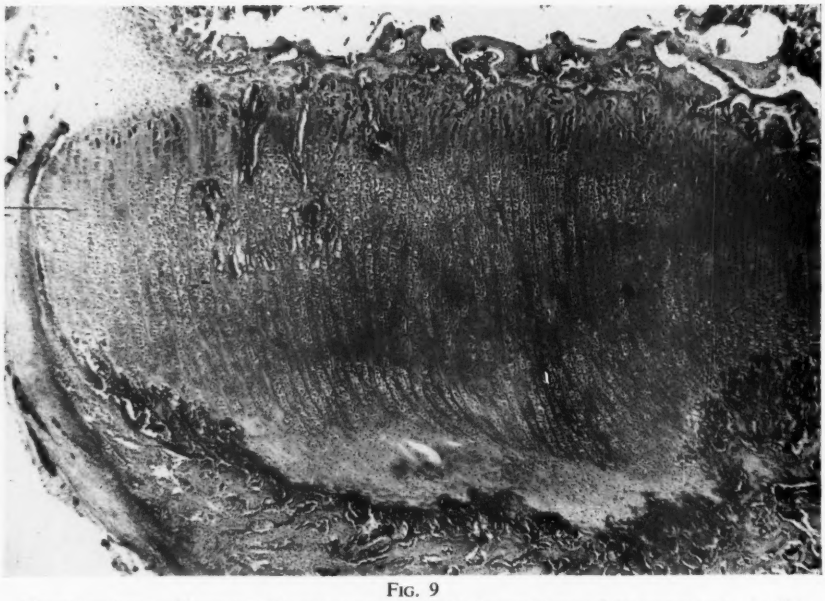


FIG. 9
Curved columns of hypertrophic cells convex towards the periphery of the metaphysis. (×60.)

affected the epiphysial side of the plate even more than the metaphysial side. These areas of epiphysial damage included severe necrotic changes of the epiphysial trabeculae and particularly of the bone plate (Fig. 11). The osteocytes of these areas were dead or showed clear signs of degeneration, and the continuity of the bone plate, which in the normal is only interrupted by the narrow vascular canals, had large gaps through which profuse vessels penetrated deep into the growth cartilage proper (Fig. 12). In all cases free from degeneration, irrespective of the thickness of the growth cartilage, the germinal and proliferative layers of the columns were completely normal, both in their staining and reproductive ability. It was also noted that while the metaphysial side of the epiphysial cartilage always became convex, the bone plate covering the growth cartilage at its epiphysial end usually remained flat.

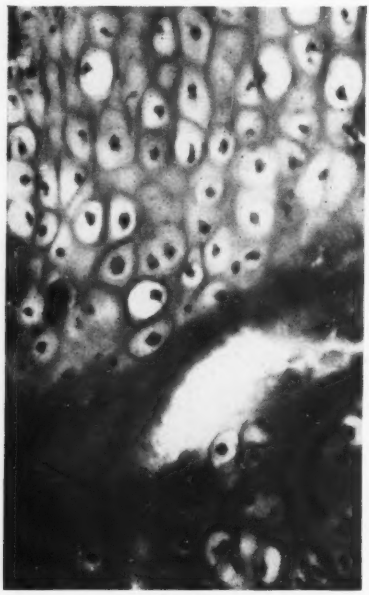


FIG. 10
Hypertrophic cells are still visible beyond the fibrous band. (×320.)

When finally the vascular penetration gave place to the formation of a bone bar, it was always after the vascular progression from both ends had bridged the height of the epiphysial cartilage.

Histological and vascular changes after removal of the compressing clamps—This study was based on the nine rabbits which were allowed to survive for from two to seventy-five days after the clamp had been in position for twelve, fifteen or twenty-one days.

The most striking feature in most of the animals was the rapidity of calcification-ossification after removal of the clamp. The columns became incorporated with the primitive bone (Fig. 13)

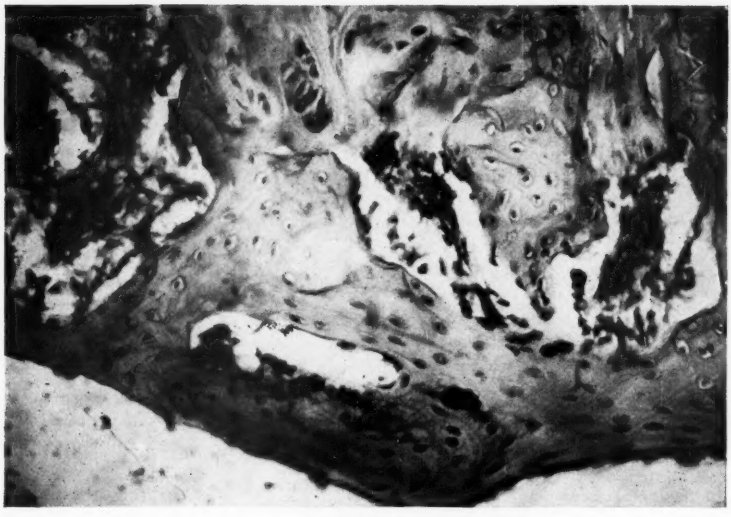


FIG. 11
Severe damage to the bone plate covering the epiphysial cartilage, with profuse vascular invasion. (×250.)

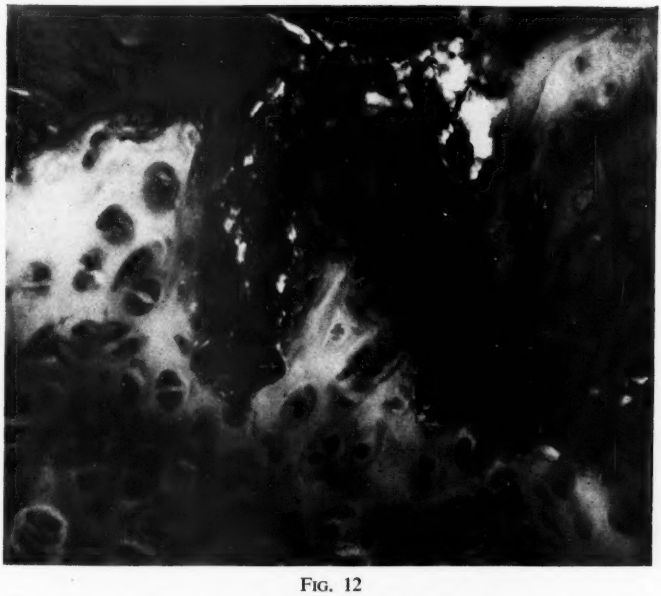


FIG. 12
The vascular invasion across the dead bone plate reaches the area of germinal layer. (×400.)

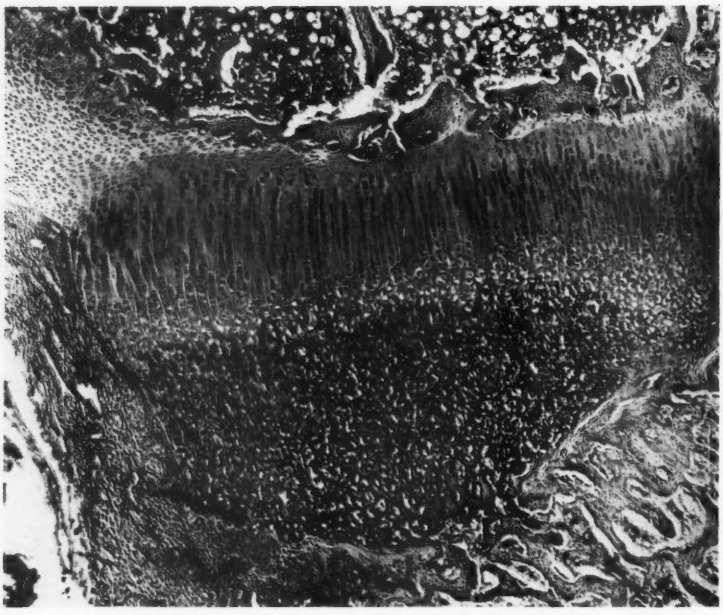


FIG. 13
Rapid ossification of the columns of hypertrophic cells after removal of compression. (×70.)

FIG. 14
Great vascular activity at the metaphysial side of the growth cartilage. (×300.)

FIG. 15

The fibrous band at the end of the columns of hypertrophic cells remains free for some time from being the site of preliminary bone. (×700.)

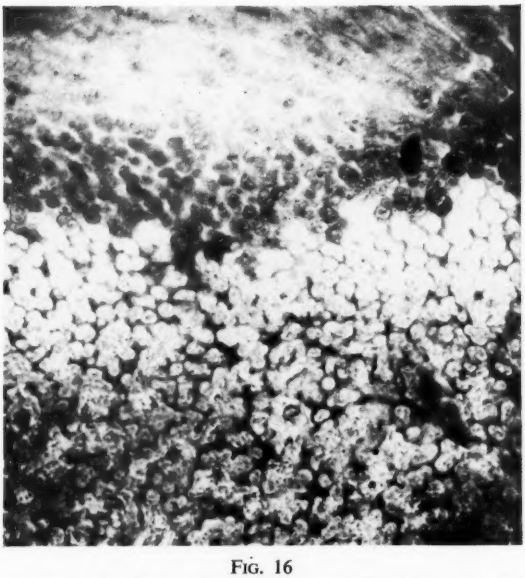


FIG. 16

Cell capsules in the process of being converted into bone. (×160.)

precisely up to their normal level for the upper tibial growth plate; that is, leaving the first eight to twelve hypertrophic cells untouched. In some sections of well injected material, we have been able to witness the rapid progress of the vessel loops back towards their normal level in the growth cartilage (Fig. 14). The rate of vascular progress could be better appreciated in those rabbits in which progressive flexion of the knee caused a steady displacement of the cone of greatest pressure towards the back of the growth cartilage, situated anteriorly to the zone of greatest pressure at the time the animal was destroyed; this showed how the vessels progressed towards their normal place of occupation. One circumstance was, nevertheless, indispensable for the rapid incorporation of the enormous mass of hypertrophic cells, namely, that no obvious signs of degeneration were present. When evident fibrotic or necrotic changes of the cells were observed, the affected area remained free for some time from being the seat of preliminary bone (Fig. 15).

The pattern of the new bone was always the same (Fig. 16). Small irregular spaces, enlarged remains of the cell capsules, were surrounded by newly calcified cartilage without, as yet, any osteoblasts and even less osteocytes appearing in it.

Metaphysial trabeculae adjacent to the growth cartilage were found often to have dead osteocytes. This gave evidence of the amount of compression under the growth cartilage. But in this, as well as in the animals with only mild changes in the hypertrophic layer, the limits of the enlarged growth cartilage were clearly seen, even several days after the area of hypertrophic cells had become calcified.

Growth changes after continual compression—All the rabbits had radiographs of both hind limbs taken at the beginning and end of the experiments. For this investigation only the tibia was considered suitable. In those animals in which the length of the tibia had not been altered during preparation, both tibiae were compared for length after removal of soft tissue.

FIG. 17
Deformed tibia of rabbit killed three months after the experiment.

It was found that a compression of less than seven days does not permanently affect growth. Compression for from seven to ten days had shortened by one millimetre all the tibiae except for one in which there was no alteration of length. All the tibiae subjected to compression for over twelve days had two or more millimetres of shortening, with the exception of two rabbits (numbers 12 and 38) which showed two millimetres of shortening after only seven and eight days respectively. The greatest shortening found was four millimetres after sixteen days of compression.

Several of the animals allowed to survive for varying durations after removal of the clamp showed increasing deformity, particularly of the tibia. The deformity varied, but flexion was the most frequent; this was due to the growth of the anterior part of the epiphysial cartilage which still remained active when the posterior side was not growing any more. It was rare to find varus or valgus deformity from unbalanced growth (Fig. 17). In some animals fine metal markers in the bone aided detection of changes in growth responsible both for the shortening and the deformity.

The study of the vascular pattern by the perfusion method described earlier, showed that the vascularity at the metaphysial end of the growth cartilage was prevented from progressing along the columns of hypertrophic cells while it remained normal at its epiphysial side (Fig. 18).

In all growth cartilages which showed degeneration the epiphysial side of the cartilage

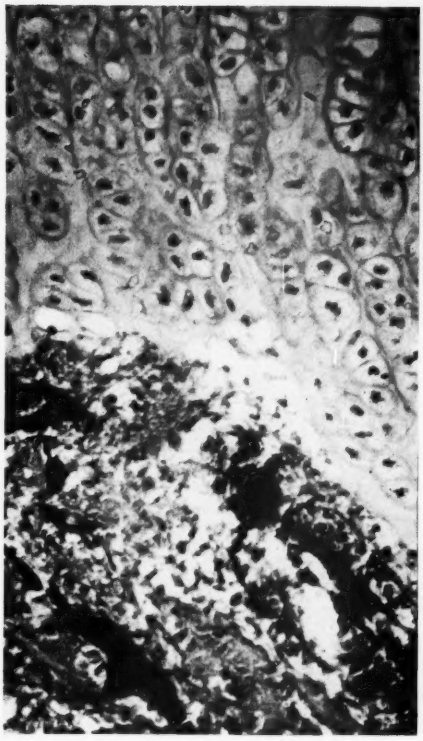


FIG. 18

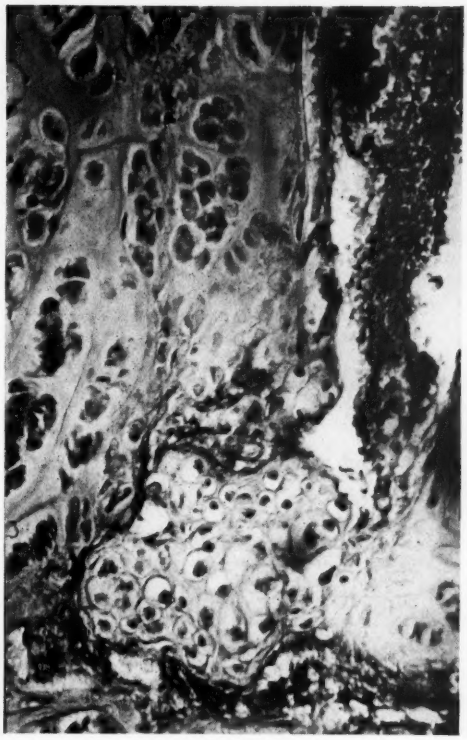


FIG. 19

Figure 18—Vessels of the metaphysial side of the growth cartilage unable to progress along the columns of hypertrophic cells. (×250.) Figure 19—Vessels from the epiphysial side of the cartilage advancing towards the depths of the columns in severe degeneration. (×250.)

showed a vascular disorder, characterised by early ischaemia followed by profuse, irregular vascular proliferation (Fig. 19).

Calcification—We had special interest in the effect of compression on calcification. We examined particularly the metaphysial side of the columns with the help of the light microscope, the electron microscope and microradiography. In all the animals in which this study was made it was found that the area of calcification remained constant at about the level reached at the time the clamps began to work. Thus, compression caused interruption of the progressive calcification which normally precedes the first phase of osteogenesis.

DISCUSSION

To emphasise the main points for discussion, it seems appropriate first to summarise our findings. The present investigation has shown that continuous severe compression of the epiphysial cartilage of the rabbit alters its normal function in the following manner.

Stage one: up to seven days of compression there is an inhibition of degeneration of the hypertrophic cells at the end of the growth columns. This allows the cells to survive for an, as yet, indefinite number of days: in some of our experiments, up to more than twelve days. During all this time cells from the proliferative section of the columns continue to develop into hypertrophic cells while the rate of cell division at the proliferative level of the columns remains apparently normal. In these circumstances the growth cartilage at the area of greatest pressure may develop to about four times its normal height.

Stage two: at approximately eight to ten days after the initiation of compression, the continuous accumulation of the new cartilage cells and their lack of subsequent removal at the metaphysial end of the epiphysial cartilage causes the compression to increase. This occurs despite the increase in the distance between the pins, because of compression of the springs of the clamp. At about ten days changes appear in the epiphysial end of the growth cartilage. From then on, the suffering of the cartilage cells and their organisation appears progressively greater until the next phase is reached.

Stage three: at about fourteen days or more the disorganisation is, in general, severe and mostly irreversible. The bone plate covering the epiphysial side of the cartilage has dead cells and appears broken into fragments. The new addition of cells by division of the proliferative section is interrupted and shortly after this, irregular vascular invasion occurs from both ends, despite the continuation of compression.

Stage four consists of the fusion of the growth cartilage by bone formation following the vascular invasion of the previous phase, beginning by establishing a narrow bone bridge across the growth plate.

It is interesting that compression has two early features, namely the " protection " of the hypertrophic cells and the lack of action upon cell reproduction in the proliferative section of the cartilage.

It is also interesting that the normal regular progression of the vascular loops at the metaphysial end of the growth plate is interrupted from the first stages of compression. Another important observation is the prevention of calcification of the matrix along the intercolumnar spaces. It appears that the first row of hypertrophic cells, not yet surrounded by a calcified matrix at the beginning of compression, remains alive until the increasing compression causes them to disintegrate in the last stages of the experiment.

The return of the growth cartilage to normal function after compression has been discontinued depends on the severity of the damage suffered by the epiphysial side of the growth cartilage. Where no serious damage has been caused to the epiphysial side of the columns, total regeneration takes place very rapidly and is always preceded by the realignment of the vessels of the metaphysial side of the cartilage. Calcification in the intercolumnar matrix up to the normal levels recurs within four days of the removal of the compression. Concomitantly, the vessels of the metaphysial side of the growth cartilage penetrate the cartilage columns to their normal level.

In the preceding paper of this series (Trueta and Amato 1960) it was reported that the interruption of the vascular supply at the metaphysial side of the growth cartilage caused calcification to cease and allowed the hypertrophic cells to survive for a long time. On the other hand, the interruption of the blood flow at the epiphysial side of the cartilage caused severe permanent damage which, on many occasions, ended by fusion of the growth plate.

The present work shows that similar changes are produced by constant pressure. This suggests that the mechanism of action is alike in both cases, namely, by interruption of blood flow.

It has been shown by the use of compression that the epiphysial blood supply is much better protected against excessive pressure than the metaphysial blood flow. Probably the solid roof constituted by the bone plate protects the net of vessels underneath it responsible for the nourishment of the germinal and proliferative section of the epiphysial cartilage.

CONCLUSIONS

From this work it may be concluded that persistent compression affects the growth plate by interference with the blood flow on one or both sides of the growth cartilage.

Despite exertion of the same pressure upon both sides of the growth plate, only the metaphysial side was readily affected in the early stages, for, as long as no damage was caused to the epiphysial side of the growth cartilage, the lesions were fully reversible.

Interference with growth was directly proportionate to the damage caused by compression to the epiphysial side of the growth plate and, in general, to the duration of compression.

The first signs of interference with the metaphysial side of the plate were the lack of vascular progression and concomitant retardation of calcification.

When severe degeneration was not present the growth cartilage recovered within four days.

The matrix was ready for calcification all the time, as shown by the extremely rapid calcification occurring soon after the compression had ceased and the vessels were able to reach their proper place.

It seems justified to believe that the first hypertrophic cell not to be calcified after removal of the clamp is the one around which the matrix has not yet changed sufficiently to have an affinity for the apatite crystals. As in moderate compression, the division of the proliferative cells continues and it seems it must be the age, or even more likely the distance from the transudate coming from the epiphysial side of the growth cartilage that conditions the maturity of the cell, which prepares the field for calcification and thus initiates the osteogenic process.

Views similar to this have been advanced by Ham (1957) and his school.

REFERENCES

HAM, A. W. (1957): *Histology*. Third edition. London: Pitman Medical Publishing Co. Ltd.

TRIAS, A. (1961): Effect of Persistent Pressure on the Articular Cartilage. An Experimental Study. *Journal of Bone and Joint Surgery*, **43–B,** 376.

TRUETA, J., and AMATO, V. P. (1960): The Vascular Contribution to Osteogenesis: III. Changes in the Growth Cartilage Caused by Experimentally Induced Ischaemia. *Journal of Bone and Joint Surgery*, **42–B,** 571.

TRUETA, J., and HARRISON, M. H. M. (1953): The Normal Vascular Anatomy of the Femoral Head in Adult Man. *Journal of Bone and Joint Surgery*, **35–B,** 442.

TRUETA, J., and MORGAN, J. D. (1960): The Vascular Contribution to Osteogenesis. I. Studies by the Injection Method. *Journal of Bone and Joint Surgery*, **42–B,** 97.

STUDIES IN THE TRANSPLANTATION OF BONE

I. Assessment of Antigenicity. Serological Studies

R. GEOFFREY BURWELL, LEEDS and G. GOWLAND, LONDON, ENGLAND

From the Departments of Anatomy and Bacteriology, University of Leeds

The introduction of the bone bank in 1942 by Inclán, and particularly the use of freeze-dried bone (Kreuz, Hyatt, Turner and Bassett 1951) has been followed by encouraging clinical reports of the method (Carr and Hyatt 1955; Berkin, Yeoman, Williamson, Zinnemann and Dexter 1957). However, it is the experience of most orthopaedic surgeons that, although there is a place for the stored bone graft, cancellous bone obtained from the patient's own iliac crest may provide a more satisfactory material for grafting (Carnesale and Spankus 1959). The inferior results obtained with stored bone are possibly due, not to the process of preparation of the bone, but to an incompatibility between the tissues of the host and the graft.

Much of the fundamental work on transplantation immunity has been concerned with skin homografts (Medawar 1958; Brent 1958; Lawrence 1959) and less work has been done on homografts of bone. Five methods are available for the assessment of the antigenicity of a bone homograft. 1) Serological methods have been utilised to try to demonstrate the formation of circulating antibodies after the insertion of homografts of bone and after injections of extracts of bone (Bonfiglio, Jeter and Smith 1955; Curtiss and Herndon 1955, 1956; Curtiss, Powell and Herndon 1959). 2) The inflammatory response surrounding fresh homografts of bone has been used to assess the degree of immunological reaction of the host against the graft (Bonfiglio *et al.* 1955; Curtiss, Chase and Herndon 1956; Enneking 1957; Enneking, Gratch and Ethridge 1957; Enneking and Gratch 1959; Heslop, Zeiss and Nisbet 1960). 3) Skin homografts are more rapidly rejected if the animal has been sensitised previously by the transplantation of certain tissues from the same donor. Chalmers (1959) has utilised this fact to establish the degree of immunity developing after primary homografts of fresh and freeze-dried bone. 4) The production of immune cells in the lymph nodes draining the areas of implantation of fresh homografts of cancellous bone has been investigated by the writers and will form the basis of a further communication. 5) The induction of a state of immunological tolerance to bone has recently been reported (Nisbet, Heslop and Zeiss 1960). Tolerance might prove to be a valuable experimental indicator for assessment of the antigenicity of bone.

The present communication describes a series of investigations in rabbits and mice designed to demonstrate the production of circulating antibodies formed in response to bone homografts and to extracts of homologous and heterologous bone.

MATERIALS AND METHODS

Electrophoretic studies—primary and secondary bone homografts—Under general anaesthesia induced by intravenous sodium pentobarbitone, one ilium was removed from each of two pairs of adult male rabbits obtained from different litters. The cortical bone was removed from one face of the excised ilium and a graft of cancellous bone one centimetre square was prepared. Each graft was inserted into the gluteus maximus muscle of its partner. Three weeks later secondary grafts of a similar nature were exchanged between each pair, but into the opposite gluteus maximus. Blood samples were obtained from the marginal vein of the ear of all four animals one, two and three weeks after the first, and one and two weeks after the second operation. The sera from these animals were analysed by electrophoresis.

Immunological studies—bone homografts and autografts—Cancellous bone was obtained from the ilia of four adult male albino rabbits. Under intravenous sodium pentobarbitone anaesthesia, one ilium was

excised, the outer cortex removed and small blocks of cancellous bone weighing 95 to 100 milligrams were prepared. Each graft was inserted into the subcutaneous tissue on the ventral aspect of one ear one to two centimetres from the external auditory meatus in twenty-seven adult male rabbits (Flemish giants). These rabbits were bled from the marginal vein of the ear immediately before killing at intervals of two (four animals), four (five animals), five (two animals), six (four animals), eight (two animals), ten (five animals), twelve (one animal) and fourteen (four animals) days after operation. In three additional rabbits a secondary bone graft was inserted in close proximity to the first graft three weeks after the first operation; blood was obtained from these animals before killing at four, eight and fourteen days after the insertion of the second graft.

Controls were provided by twelve adult male Flemish giants in which blocks of cancellous bone weighing 95 to 100 milligrams obtained from the animals' own ilia were inserted into the subcutaneous tissue of the ear. The animals were bled before killing at two (two animals), four (three animals), six (three animals), eight (two animals), ten (one animal) and fourteen (one animal) days after grafting. The effect of secondary autogenous grafts inserted three weeks after the first operation was investigated in three further rabbits; blood samples were taken just before killing at four, eight and fourteen days after grafting. The autografted animals served not only as controls, but also to exclude the production of auto-antibodies to traumatised bone.

At the end of each experiment, blood was taken from the homograft and the autograft donors, stored in modified Alsever's solution (Alsever and Ainslie 1941), and later used to provide donor red blood cells for the detection of iso-haemagglutinins. Donor cancellous bone was also obtained from ilia, upper femora and vertebrae for the preparation of extracts.

The rabbits in both the homograft and autograft groups were also used for a histological examination of the reactivity of the regional lymph nodes to grafts of fresh bone.

Immunological studies—homologous bone extracts—An attempt was made to immunise four rabbits with saline extracts of bone obtained from the four donor animals. One extract from each donor was used to immunise one rabbit. Injections were carried out over a period of eighty-five days and extract equivalent to 2·5 grammes of bone was used. For the first two injections the extract was given subcutaneously in an oil emulsion,* the later injections being given intravenously. Ten days after the last injection blood was obtained for the immunological examinations.

Immunological studies—heterologous bone extracts—A saline extract of the bone from one rabbit was used to immunise mice. Ten adult male albino mice weighing between 18 and 22 grammes were given a course of five 0·1 millilitre injections of bone extract by the intraperitoneal route at intervals of two days. Twelve days after the last injection blood was obtained from all the mice. The sera were separated and then pooled before immunological analysis.

Serum samples—The blood was allowed to clot and the serum, separated by centrifuging, was stored in sterile rubber-stoppered glass tubes at 4 degrees Centigrade until required. The sera were examined for iso-haemagglutinins formed in response to the respective donor of homograft or autograft bone, precipitins and also passive haemagglutinins to protein or polysaccharide components in the bone.

Electrophoresis was carried out on filter paper soaked in " Oxoid " barbitone buffer (pH=8·5) using a standard " Eel " electrophoresis apparatus.

Preparation of bone extracts—Cancellous bone obtained from the homograft donors and all the autografted rabbits was used to prepare the bone extracts. The bone was cut up into small fragments, weighed, frozen in solid carbon dioxide and crushed with fragments of solid carbon dioxide in a previously cooled mortar. Cold 0·85 per cent saline was then added in the ratio of 1·0 millilitre to every 500 milligrams of bone. The resulting suspension of bone was placed in rubber-stoppered glass tubes which were kept at 4 degrees Centigrade for seven days, the suspensions being shaken for periods of ten minutes four times daily. The suspensions were then centrifuged at 3,000 r.p.m. for thirty minutes at 4 degrees Centigrade and the supernatant extract was decanted and stored at 4 degrees Centigrade.

Serological techniques—*Haemagglutinins* were estimated by adding 0·5 millilitre of a 2 per cent saline suspension of washed donor red blood cells to 0·5 millilitre amounts of a series of two-fold dilutions of the test serum in saline. After being shaken, the mixtures were kept at 37 degrees Centigrade in a water bath for one and a half hours and read after standing at room temperature overnight.

Precipitins—Three types of precipitin tests were carried out: 1) Diffusion in agar using the " punch hole " technique. The tests were read after fourteen days. 2) Diffusion in agar columns (Oakley and Fulthorpe 1953). The tests were read after fourteen days. 3) By placing extracts on top of the test serum in a capillary tube. Tests were kept for five hours and read at half-hour intervals.

*The oil base consisted of 1 part (w/v) lanoline and 15 parts (v/v) of liquid paraffin.

Passive haemagglutinins—1) Agglutination of tanned sheep erythrocytes sensitised to proteins in the bone extracts (Boyden 1951). Sheep blood was obtained from the local abattoir and preserved in modified Alsever's solution at 4 degrees Centigrade. Cells were always aged for seven days before use. Tanned sheep red cells were sensitised with various amounts of bone extract. To 0·5 millilitre amounts of serial dilutions of the serum under test was added 0·05 millilitre of a 2·5 per cent suspension of tanned sensitised sheep erythrocytes. The tests were read after overnight incubation at room temperature. The diluent used was always 1:100 normal rabbit serum in saline. 2) Agglutination of sheep erythrocytes sensitised to polysaccharides in the bone extracts (Chen 1952). Attempts were made to sensitise sheep erythrocytes to any compounds of a polysaccharide nature present in the bone extracts. One volume of washed, packed sheep cells was added to nine volumes of various concentrations of the bone extract, incubated for two hours at room temperature, washed three times in saline and resuspended in saline to give a 2·5 per cent suspension. To 0·5 millilitre amounts of serial dilutions of the test sera (starting at a dilution of a half) were added 0·05 millilitre of sensitised cells. Tests were again read after standing at room temperature overnight for both macroscopic and microscopic agglutination.

RESULTS

Electrophoretic fractionation of the serum of the four rabbits receiving primary and secondary homografts of bone did not reveal a change in any component of the serum.

The immunological techniques used failed to detect the presence of any circulating classical antibodies formed in response to the homografts and the autografts of cancellous bone.

It was not possible to demonstrate any humoral antibody in rabbits which had received multiple injections of a saline extract of bone.

The injections of saline extract of rabbit bone into mice did however evoke the production of classical antibodies. The mouse sera gave a positive precipitin test when in contact with the heterologous extract, a positive haemagglutination at a dilution of 1:160 with tanned sheep erythrocytes sensitised to proteins in the bone extract and they agglutinated sheep erythrocytes sensitised to polysaccharides in the extract of bone at a dilution of 1:40.

DISCUSSION

The failure to demonstrate the presence of iso-haemagglutinins in rabbits receiving homografts of cancellous bone is consistent with the findings of Medawar (1946), who was unable to demonstrate the production of red-cell iso-agglutinins after massive skin homotransplantation in rabbits. Recently, however, serum iso-agglutinins to both red blood cells and leucocytes have been shown to occur after skin homografting in mice (Amos, Gorer, Mikulska, Billingham and Sparrow 1954).

That haemagglutinating antibodies are formed in response to large bone transplants was shown by Curtiss and Herndon (1955, 1956) who transplanted the knee joint of group "A"-positive dogs into five group "A"-negative dogs, and showed that in two animals specific antibody against the blood group "A" substance had been produced. These workers also demonstrated a significant rise in the iso-agglutinin titres against dog erythrocytes in rabbits receiving heterologous transplants from a dog of whole ilium, minced and washed ilium or marrow-free cortical bone. They suggested that the agglutinating antibody might have developed as the result of an antigenic stimulus present in the transplanted bone itself. In this connection, Weinberg, Makin, Nelken and Gurevitch (1959) have shown that blood group "A" and " B " antigens are present in human cancellous bone.

The detection of classical antibodies after homografting may be difficult because the antibodies are adsorbed either by the antigens in the donor tissue (Feldman and Yaffe 1958), or by common antigens present in the tissue cells of the recipient (Merrill 1959). It is possible then that humoral antibodies were formed in response to the bone homografts used in our investigations, but in amounts which did not reach detectable levels. Small amounts of haemagglutinins may have been produced to both rabbit and blood group antigens (Nelken, Birnbaum and Gurevitch 1960) present in the bone marrow and also possibly within the bone tissue itself (Weinberg and colleagues 1959).

The inability to reveal precipitins in the sera of the writers' experimental animals receiving bone homografts is in agreement with the results of Bonfiglio and colleagues (1955), who were unable to show the presence of precipitins after primary and secondary homografts of bone to the ulna in the rabbit. The formation of precipitins to primary skin homografts in the rabbit has been recorded (Bollag 1956), but this work has not been confirmed (Merrill 1959). Multiple skin homografts, however, have been reported to produce positive precipitin tests in 50 per cent of rabbits (Merrill 1959).

In our experiments the use of sheep red blood cells on to which any possible antigen present in the bone extracts might be adsorbed, did not reveal the formation of passive haemagglutinins to either protein or polysaccharide components of the bone. It is not surprising therefore, with the failure to demonstrate serum haemagglutinins, precipitins or passive haemagglutinins, that no disturbance in the electrophoretic pattern of the serum of the homografted animals was detected.

The effect of transplants of bone upon the level of the serum protein properdin has recently been studied by Curtiss, Powell and Herndon (1959). They did not find any significant alteration in properdin titres in rabbits receiving homografts of cancellous bone. These observations are in accord with work which has shown that the survival of primary and secondary homografts of skin is not effected by an artificial lowering of the serum properdin levels (Hubay, Wityk and Holden 1958).

Injections of extracts of homologous bone in our rabbits failed to produce demonstrable circulating antibodies, a finding in keeping with the results of Allgöwer, Blocker and Engley (1952) working with skin, and Bonfiglio and his colleagues (1955) using a soluble fraction of homologous bone injected into rabbits. The latter workers did however show that when an extract (with adjuvants) of the pooled bone from eight donors was injected, a low titre of a complement-fixing antibody was demonstrable. The effect of injections of the insoluble as well as the soluble fraction of bone upon antibody production has been studied in rabbits by Curtiss and his colleagues (1959). They were unable to reveal the production of either haemagglutinins, precipitins or any increased binding by bone of radio-iodinated gamma globulin obtained from the sera of their animals. Extracts of heterologous skin have, however, produced a recognisable titre of cytotoxic antibodies and precipitins after injection into the rabbit (Allgöwer and colleagues 1952). Merrill (1959) has also shown that the subcutaneous injection of an extract of human leucocytes into rabbits produced cytotoxins and precipitins. Siegrist and Enneking (1960), however, failed to demonstrate serum antibodies formed in response to heterologous extracts of rat bone injected into hamsters, a failure which they attributed to an error in the method of preparation of their bone extract. Aoike and Iiji (1960) also were unable to reveal the definite formation of serum antibodies in rabbits receiving a saline extract of ox bone. Our experiments, in which an extract of rabbit bone was injected into mice and caused the production of both precipitins and passive haemagglutinins, is the first demonstration of the formation of serum antibodies to injections of heterologous extracts of bone. It is possible that the technique of heterologous injections would provide a useful method whereby certain other antigenic components of bone might be revealed.

Several workers have searched for serum antibodies formed in response to fresh tissue heterografts of bone. Curtiss and Herndon (1955, 1956) have demonstrated the production of iso-agglutinins against dog erythrocytes in rabbits having received transplants from a dog of whole ilium, minced and washed ilium or marrow-free cortical bone. Siegrist and Enneking (1960), Aoike and Iiji (1960) and Kawamura, Hosono, Takahashi, Tomita, Nakazoe, Hirayama and Kido (1960) were unable to find any evidence for the production of classical antibodies to the bone heterografts used in their animal investigations. However, Aoike and Iiji did show that, in one patient who had received an ox bone graft, immune products were detected by the complement fixation test and in this patient an allergic response to an intracutaneous injection of ox osseo-mucoid was also demonstrated.

Though under certain conditions humoral antibodies are formed in response to transplants or extracts of both skin and bone, there is, however, evidence to suggest that serum antibodies are not the instruments of transplantation immunity (Medawar 1959).

SUMMARY

1. An immunological examination of the sera of thirty rabbits which had received primary and secondary homografts of cancellous bone into a subcutaneous site did not reveal the presence of circulating precipitins, haemagglutinins or passive haemagglutinins. These findings are consistent with the observations of Bonfiglio and his colleagues (1955).

2. Electrophoretic examination of the serum of four rabbits receiving primary and secondary homografts of bone into an intramuscular site did not reveal any change in the serum protein fractions.

3. A search for auto-antibodies produced by primary and secondary autografts of cancellous bone was unsuccessful in fifteen rabbits.

4. The multiple injections of saline extracts of bone into four rabbits did not evoke the production of demonstrable circulating antibodies, results which are in accord with the findings of Bonfiglio and colleagues (1955) and Curtiss and colleagues (1959).

5. For the first time the production of classical antibodies in response to injections of extracts of heterologous bone has been recorded. The repeated injections of a saline extract of rabbit bone intraperitoneally into ten mice produced demonstrable precipitins and passive haemagglutinins both to protein and polysaccharide fractions present in the bone extracts.

6. Knowledge concerning the production of humoral antibodies to transplants and extracts of bone has been reviewed.

The writers are indebted to Professor A. Durward and Professor C. L. Oakley, F.R.S., for the facilities which were unsparingly provided in their Departments. Dr W. N. Anderson of the Department of Chemical Pathology undertook the electrophoretic fractionation, for which we are most grateful. Our thanks are due to Professor P. B. Medawar, F.R.S., for helpful criticism and for reading the manuscript.

REFERENCES

ALLGÖWER, M., BLOCKER, T. G., Jun., and ENGLEY, B. W. D. (1952): Some Immunological Aspects of Auto- and Homografts in Rabbits, Tested by *in vivo* and *in vitro* Techniques. *Plastic and Reconstructive Surgery*, **9**, 1.

ALSEVER, J. B., and AINSLIE, R. B. (1941): A New Method for the Preparation of Dilute Blood Plasma and the Operation of a Complete Transfusion Service. *New York State Journal of Medicine*, **41**, 126.

AMOS, D. B., GORER, P. A., MIKULSKA, B. M., BILLINGHAM, R. E., and SPARROW, E. M. (1954): An Antibody Response to Skin Homografts in Mice. *British Journal of Experimental Pathology*, **35**, 203.

AOIKE, and IIJI (1960): The Development of Immune Products in the Rabbit after Ox-bone Grafting. *Journal of Bone and Joint Surgery*, **42–A**, 543.

BERKIN, C. R., YEOMAN, P. M., WILLIAMSON, G. M., ZINNEMANN, K., and DEXTER, F. (1957): Freeze-dried Bone-grafts. *Lancet*, **i**, 730.

BOLLAG, W. (1956): Serologischer Nachweis von individualspezifischen Gewebsantikörpern. Beziehungen zum Problem der Organtransplantationen. *Schweizerische Medizinische Wochenschrift*, **86**, 687.

BONFIGLIO, M., JETER, W. S., and SMITH, C. L. (1955): The Immune Concept: its Relation to Bone Transplantation. *Annals of the New York Academy of Sciences*, **59**, 417.

BOYDEN, S. V. (1951): The Absorption of Proteins on Erythrocytes Treated with Tannic Acid and Subsequent Haemagglutination by Antiprotein Sera. *Journal of Experimental Medicine*, **93**, 107.

BRENT, L. (1958): Tissue Transplantation Immunity. *Progress in Allergy*, **5**, 271.

CARNESALE, P. L., and SPANKUS, J. D. (1959): A Clinical Comparative Study of Autogenous and Homogenous Bone Grafts. *Journal of Bone and Joint Surgery*, **41–A**, 887.

CARR, C. R., and HYATT, G. W. (1955): Clinical Evaluation of Freeze-dried Bone Grafts. *Journal of Bone and Joint Surgery*, **37–A**, 549.

CHALMERS, J. (1959): Transplantation Immunity in Bone Homografting. *Journal of Bone and Joint Surgery*, **41–B**, 160.

CHEN, T. H. (1952): Studies on Immunization Against Plague. IV. The Method of the Haemagglutination Test and Some Observations on the Antigen. *Journal of Immunology*, **69**, 587.

CURTISS, P. H., Jun., CHASE, S. W., and HERNDON, C. H. (1956): Immunological Factors in Homogenous Bone Transplantation. II. Histological Studies. *Journal of Bone and Joint Surgery*, **38–A,** 324.

CURTISS, P. H., Jun., and HERNDON, C. H. (1955): Immunological Factors in Homogenous Bone Transplantation. I. Serological Studies. *Annals of the New York Academy of Sciences*, **59,** 434.

CURTISS, P. H., Jun., and HERNDON, C. H. (1956): Immunological Factors in Homogenous Bone Transplantation. I. Serological Studies. *Journal of Bone and Joint Surgery*, **38–A,** 103.

CURTISS, P. H., Jun., POWELL, A. E., and HERNDON, C. H. (1959): Immunological Factors in Homogenous Bone Transplantation. III. The Inability of Homogenous Rabbit Bone to Induce Circulating Antibodies in Rabbits. *Journal of Bone and Joint Surgery*, **41–A,** 1,482.

ENNEKING, W. F. (1957): Histological Investigation of Bone Transplants in Immunologically Prepared Animals. *Journal of Bone and Joint Surgery*, **39–A,** 597.

ENNEKING, W. F., and GRATCH, A. (1959): The Effect of Total Body Irradiation on Bone Transplants in Parabiosed Animals. *Journal of Bone and Joint Surgery*, **41–A,** 463.

ENNEKING, W. F., GRATCH, A., and ETHRIDGE, H. (1957): The Effect of Total Body Irradiation on Bone Transplants in Parabiosed Animals. *Surgical Forum*, **8,** 561.

FELDMAN, M., and YAFFE, D. (1958): Immunogenetic Studies on X-irradiated Mice Treated with Homologous Hematopoietic Cells. *Journal of the National Cancer Institute*, **21,** 697.

HESLOP, B. F., ZEISS, I. M., and NISBET, N. W. (1960): Studies on Transference of Bone. I. A Comparison of Autologous and Homologous Bone Implants with Reference to Osteocyte Survival, Osteogenesis and Host Reaction. *British Journal of Experimental Pathology*, **41,** 269.

HUBAY, C. A., WITYK, J. J., and HOLDEN, W. D. (1958): The Effect of the Properdin System upon First and Second Set Homografts. *Surgery, Gynecology and Obstetrics*, **107,** 311.

INCLÁN, A. (1942): The Use of Preserved Bone Graft in Orthopaedic Surgery. *Journal of Bone and Joint Surgery*, **24,** 81.

KAWAMURA, HOSONO, TAKAHASHI, TOMITA, NAKAZOE, HIRAYAMA and KIDO (1960): Experimental Studies of Heterogenous-Bone Transplantation. *Journal of Bone and Joint Surgery*, **42–A,** 543.

KREUZ, F. P., HYATT, G. W., TURNER, T. C., and BASSETT, A. L. (1951): The Preservation and Clinical Use of Freeze-dried Bone. *Journal of Bone and Joint Surgery*, **33–A,** 863.

LAWRENCE, H. S. (1959): Homograft Sensitivity. An Expression of the Immunologic Origins and Consequences of Individuality. *Physiological Reviews*, **39,** 811.

MEDAWAR, P. B. (1946): Immunity to Homologous Grafted Skin. II. The Relationship Between the Antigens of Blood and Skin. *British Journal of Experimental Pathology*, **27,** 15.

MEDAWAR, P. B. (1958): The Homograft Reaction. *Proceedings of the Royal Society of London*, **149–B,** 145.

MEDAWAR, P. B. (1959): In *Cellular and Humoral Aspects of the Hypersensitive States*, Chapter 14. Edited by H. S. Lawrence. London: Cassell.

MERRILL, J. P. (1959): Antigen and Antibody in Transplantation Immunity: Some Problems of Investigation. In *Biological Problems of Grafting*, p. 34. Edited by F. Albert and P. B. Medawar. Oxford: Blackwell Scientific Publications.

NELKEN, D., BIRNBAUM, D., and GUREVITCH, J. (1960): Blood Groups in Rabbits. *Nature*, **186,** 321.

NISBET, N. W., HESLOP, B. F., and ZEISS, I. M. (1960): Studies on Transference of Bone. III. Manifestations of Immunological Tolerance to Implants of Homologous Cortical Bone in Rats. *British Journal of Experimental Pathology*, **41,** 444.

OAKLEY, C. L., and FULTHORPE, A. J. (1953): Antigenic Analysis By Diffusion. *Journal of Pathology and Bacteriology*, **65,** 49.

SIEGRIST, W. H., and ENNEKING, W. F. (1960): Immunologic and Histologic Investigation of Heterogenous Bone Transplants. *Journal of Bone and Joint Surgery*, **42–A,** 539.

WEINBERG, H., MAKIN, M., NELKEN, D., and GUREVITCH, J. (1959): A and B Antigens in Human Bone Tissue. *Journal of Bone and Joint Surgery*, **41–B,** 151.

STUDIES IN THE TRANSPLANTATION OF BONE

II. The Changes Occurring in the Lymphoid Tissue after Homografts and Autografts of Fresh Cancellous Bone

R. GEOFFREY BURWELL, LEEDS and G. GOWLAND, LONDON, ENGLAND

From the Departments of Anatomy and Bacteriology, University of Leeds

In an earlier paper (Burwell and Gowland 1961) it was shown that under certain conditions serum antibodies were formed in response to transplants and extracts of bone. However, there is evidence to suggest that the instruments of transplantation immunity are not serum antibodies but antibodies bound to cells of the lymphoid series (Medawar 1959*a*).

This paper is concerned particularly with a study of the changes occurring in the first regional lymph nodes draining the areas of insertion of homografts and autografts of fresh cancellous bone, but observations are also reported on lymphoid tissue at other sites.

The first histological report concerning the production of " activated cells " in regional lymph nodes draining the areas of insertion of homografts was made by Gallone, Radici and Riquier (1952), who found an increase in the pyronin-staining elements in lymph nodes draining skin homografts. Scothorne and McGregor (1955), who confirmed and extended these observations, showed that accumulations of " large lymphoid cells " occurred in the cortex, and to a lesser extent in the medulla, of the first regional nodes draining homografts of skin. They found only a weak response in the second regional nodes away from the grafts and no histological change in the spleens of their animals. They concluded that their histological findings were in keeping with experimental work which had shown that the production of immune lymphoid cells to tissue homografts was principally a regional process (Mitchison 1954; Billingham, Brent and Medawar 1954). Radici and Piredda (1957), also using rabbits, described how five days after the insertion of skin homografts the reticular cells of the medullary cords of the first regional nodes were in large part replaced by pyroninophilic cells. The regional lymph node response to skin heterografts transplanted from rabbits to rats has been described by Egdahl, Varco and Good (1958). The reaction of the first regional lymph nodes to homografts of cartilage has been studied by Craigmyle (1958), who did not find any increase in the number of "large lymphoid cells" in the nodes draining the sites of implantation. However, the insertion of secondary homografts or primary heterografts of cartilage produced in most cases the characteristic cellular response. Craigmyle concluded that cartilage is only mildly antigenic, which is consistent with the long survival time of cartilage homografts in man (Peer 1954).

The present investigations were undertaken as part of a series of studies concerned with the search for a bone material which would be more acceptable to the patient's tissues than the stored bones which are at present used in clinical bone grafting. The better results obtained by using autogenous rather than preserved homogenous bone (Carnesale and Spankus 1959) may be due in the widest sense to an " incompatibility " between the homograft and the patient's tissues. In recent years, though experimental work has shown that the transplantation of certain tissues, and particularly of skin, between members of the same species results in the production of a state of actively acquired immunity in the host (Medawar 1958), there is little information concerning the antigenicity of preserved bone (Burwell 1960*b*).

It was decided therefore to study the effect of homografts of fresh cancellous bone upon the regional lymph nodes to see whether there was evidence of an immune reaction. It is shown not only that a " large lymphoid cell " response occurs, but that this cellular reaction is sectorally distributed within the lymph node. A quantitative analysis of this " large lymphoid cell " response is also reported. Preliminary reports of this work have already appeared (Burwell 1960*a*, Burwell and Gowland 1960).

MATERIALS AND METHODS

Seventy-five adult male rabbits weighing 2·2 to 4·3 kilograms were used in the investigation. The five rabbits serving as donors of bone were of Albino strain. The recipient animals used for both homografts and autografts were Flemish Giants. In some of the animals blood samples were obtained before death and examined immunologically (Burwell and Gowland 1961).

Operative procedures—Bone was obtained from the donor animals after anaesthesia by intravenous injection of sodium pentobarbitone. The ilium was excised, the outer cortex removed and small blocks of cancellous bone weighing 95 to 100 milligrams were prepared. The recipient animals were anaesthetised by the injection of sodium pentobarbitone into the marginal vein of the right ear. Each bone graft was inserted, in fifty-one rabbits, into the subcutaneous tissue on the ventral aspect of the left ear one to two centimetres from the external auditory meatus, the wound edges being carefully apposed by means of one or two sutures of braided nylon (Fig. 1). The animals were killed two, four, five, six, eight, ten, twelve, fourteen or twenty-one days after grafting.

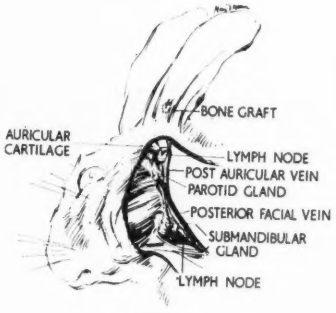


FIG. 1

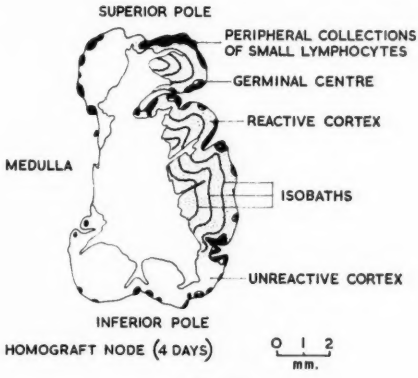


FIG. 2

Figure 1—Drawing of a rabbit's head showing the grafting site and the positions of the first (upper) and second (lower) regional lymph nodes. Figure 2—Camera lucida tracing of a section from a lymph node removed four days after the insertion of a homograft of cancellous bone into its drainage area. The distribution of the large and medium lymphoid cells in the cortex is indicated by the stippling; this is called the reactive cortex. The cortex which is unreactive is not stippled. The method of sampling the large and medium lymphoid cell content of the reactive cortex is shown by the " isobaths "—see text.

In the nineteen rabbits receiving autografts, bone from the animal's own ilium was prepared in a similar manner, and likewise inserted into the tissues of the left ear. These animals were killed two, four, six, eight, fourteen or twenty-one days after grafting (Table I).

Histological techniques—The animals were killed by intravenous sodium pentobarbitone and the first regional lymph nodes draining the operative and control ears were removed and weighed separately. The second regional nodes draining the ears were removed, together with, in many instances, a mediastinal node and a specimen from the lung. The spleen of each animal was removed and weighed and a segment four to five millimetres thick obtained for fixation. The ear grafts were removed and fixed in 10 per cent formol-saline. Carnoy's fluid was used for fixation of the lymph nodes and spleens. The lung specimens were fixed in 10 per cent neutral formol-saline. The bone specimens were decalcified with ethylene-diamine-tetra-acetic acid (Meyer 1956). The material was dehydrated, cleared and embedded in paraffin wax, the bone grafts and lung specimens being vacuum embedded. Sections of the lymphoid material were cut at 6μ thickness. The first regional nodes were mounted in serial section in twenty-four homografted and fourteen autografted animals. All other material was cut at 8μ thickness. The sections of lymph nodes and spleens were stained with methyl green-pyronin, of the bone grafts with haematoxylin and eosin and periodic acid-Schiff, and of the lungs with haematoxylin and eosin and methyl green-pyronin.

A quantitative analysis of the large lymphoid cell response was made in the reactive parts of the cortex in twenty-four nodes draining sites of homograft insertion, in three different sections from each node, and three sections in each node studied being 350 to 1,000μ apart depending upon the

size of the node. Each count was made in a field (8×8 divisions—henceforth called a "unit area") of a graticule using 10× eyepieces and a 100× oil immersion objective. The counts were made two divisions of the graticule apart, on lines (or "isobaths") four and eight divisions from the capsule of the node (Fig. 2). Less frequently, when the reactive cortex was particularly thick, counts were also made twelve, sixteen and rarely twenty divisions from the surface of the node. The cells counted in each unit area

TABLE I

WEIGHTS OF THE FIRST REGIONAL LYMPH NODES AND OF SPLEENS

Homografted animals

Serial number	Days after grafting	Weight of node (*milligrams*)		Weight of spleen (*grammes*)
		Operated side	Control side	
55	2	100	60	0·89
73	2	115	75	1·29
77	2	95	75	0·96
86	2	100	90	3·05
132	2	240	170	—
162	2	110	80	1·18
178	2	120	120	1·25
51	4	225	175	—
56	4	170	160	1·43
74	4	290	190	1·61
79	4	95	95	0·86
80	4	145	130	1·17
87	4	90	40	1·43
133	4	360	250	2·05
140	4	320	140	1·13
153	4	340	160	1·34
88	5	230	130	1·60
110	5	180	160	1·58
134	5	195	130	1·90
141	5	320	170	2·05
154	5	190	140	1·39
185	5	240	100	1·76
57	6	270	99	—
81	6	145	85	1·50
82	6	260	115	1·26

included all cells with a pale nucleus, prominent nucleoli and a variable degree of intensely pyroninophilic cytoplasm. These cells appear to correspond to the large and medium lymphoid cells of Craigmyle (1958). The nuclei of the "medium lymphoid cells" differ from the nuclei of medium lymphocytes in that the latter stain intensely with methyl green.

RESULTS

CHANGES IN WEIGHT

The weights of the first regional lymph nodes from the operated and the control sides, together with the weights of the spleens, from the homografted and the autografted animals are shown in Tables I and II.

TABLE I—*continued*

WEIGHTS OF THE FIRST REGIONAL LYMPH NODES AND OF SPLEENS

Homografted animals

Serial number	Days after grafting	Weight of node (*milligrams*)		Weight of spleen (*grammes*)
		Operated side	Control side	
89	6	230	120	2·47
135	6	290	160	1·14
142	6	340	180	1·73
58	8	230	85	—
83	8	215	55	1·60
90	8	180	40	1·71
136	8	310	130	1·77
143	8	210	90	2·26
155	8	320	170	2·10
59	10	180	85	1·54
84	10	190	110	1·56
92	10	200	190	2·40
111	10	240	140	2·05
148	10	270	120	2·47
149	10	290	40	1·03
160	10	220	150	1·77
93	12	210	200	0·94
150	12	180	155	1·57
161	12	230	110	1·70
61	14	210	210	2·45
78	14	100	65	2·79
91	14	540	180	1·74
112	14	200	100	1·22
151	14	240	150	1·75
159	14	220	140	1·49
152	21	175	140	1·60

A t-test for differences between the homograft and the autograft nodes carried out separately for nodes obtained on two, four, six, eight, ten and fourteen days reveals a statistically significant difference only on the sixth ($0{\cdot}02<P<0{\cdot}05$) and eighth ($0{\cdot}02<P<0{\cdot}05$)

days after operation. The differences between the mean weight increments of nodes removed on the fourth ($0{\cdot}05<P<0{\cdot}1$) and tenth ($0{\cdot}1<P<0{\cdot}2$) days approaches significance and, taking into account the significance at six and eight days it seems probable that with larger numbers of observations these differences too would be significant. An overall test of the difference

TABLE II

WEIGHTS OF THE FIRST REGIONAL LYMPH NODES AND OF SPLEENS

Autografted animals

Serial number	Days after grafting	Weight of node (*milligrams*)		Weight of spleen (*grammes*)
		Operated side	Control side	
64	2	70	70	1·41
96	2	120	110	2·25
97	2	95	90	1·36
65	4	145	125	2·38
98	4	65	65	1·05
99	4	130	100	1·62
66	6	125	85	1·58
100	6	170	140	1·60
101	6	150	110	2·15
67	8	165	115	1·41
102	8	150	90	0·89
103	8	160	130	1·19
104	10	240	220	2·00
105	10	230	130	1·72
106	10	80	90	2·72
107	14	110	140	2·14
108	14	120	100	1·24
109	14	110	130	1·37
71	21	60	80	—

between the homograft nodes on the one hand and the autograft nodes on the other has also been made by combining the values of P, using the method described by Fisher (1946). This test of significance gives $P<0{\cdot}005$, showing that, overall, the difference in weight increments of the homografted and the autografted nodes is highly significant.

The mean weight increment together with the standard error of each mean of the first regional lymph nodes for each time interval after operation, are plotted for both the homografted and the autografted animals in Figure 3.

The mean weights and the standard deviations of the control nodes have been calculated to be 127 ± 47 milligrams for the homografted, and 112 ± 35 milligrams for the autografted animals; the difference between these means is not significant ($0{\cdot}1<P<0{\cdot}2$).

The mean weight of forty-six spleens from the homografted animals is $1{\cdot}65\pm0{\cdot}26$ grammes, and from the autografted rabbits is $1{\cdot}67\pm0{\cdot}24$ grammes, a difference which is not significant. To determine whether there is any statistically significant difference between the mean splenic

weights of homografted animals killed at the different time intervals, the data have been subjected to an analysis of variance. This test has revealed that there is no significant difference between the mean splenic weights of the animals killed at the different time intervals after insertion of the bone homografts into the subcutaneous tissues of the ear.

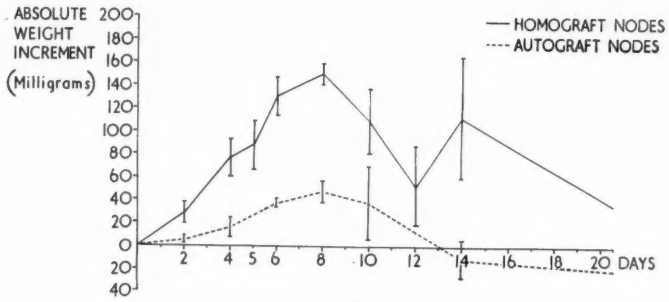


FIG. 3

Graphs to compare the mean weight changes (with standard errors) of the first regional lymph nodes draining areas of insertion of homografts with nodes draining those of autografts of cancellous bone (seventy rabbits).

HISTOLOGICAL OBSERVATIONS

The Regional Lymph Nodes

Control nodes—The cortex of the control nodes is thin in most parts and consists of germinal centres and diffuse cortical tissue (Figs. 4 and 5). The germinal centres are mainly of Flemming and transitional types, though a few primary nodules do exist. The diffuse lymphoid tissue consists mainly of small lymphocytes, though large lymphoid cells do occur sporadically throughout. The medullary trabeculae are thin and contain mature plasma cells, small lymphocytes and occasional large lymphoid cells. The sinuses of the medulla contain small lymphocytes, a few large lymphoid cells and occasional cells with pale chromatin, prominent nucleoli and a variable amount of pale pyroninophilic cytoplasm in which ingested material is sometimes seen. Adipose tissue is a frequent finding in the medullae of control nodes.

Homograft nodes—In most of the first regional lymph nodes draining sites of insertion of homografts of cancellous bone implanted two days previously, there is little change to be seen either in the germinal centres or in the diffuse lymphoid tissue of the cortex. In the nodes of animals killed four, five, six and eight days after grafting the most striking change is an increase in the thickness of the diffuse cortical tissue either on one side, or frequently towards the polar regions (Figs. 6 and 7). This thickened cortical tissue contains a great number of large lymphoid cells (Fig. 8) which are found mainly in the outer half to two-thirds of the cortex. The distribution of these large cells is not even throughout the thickened diffuse cortical tissue, and the highest concentration may be seen either to one side or towards the upper pole of the node. To these sectors of thickened diffuse cortical tissue containing great numbers of large lymphoid cells, the name reactive cortex will be applied (Fig. 9). The subcapsular regions of the reactive cortex may lack germinal centres and collections of small lymphocytes beneath the capsule are frequently found. In the unreactive parts of the cortex the diffuse lymphoid tissue is usually not thickened, does not show an increase of its large lymphoid cell content (Fig. 10) and the germinal centres are relatively large and are of Flemming or transitional type. After ten days a variation in the response of the reactive cortex is found as seen in three nodes. One node (149), still reveals sectors of the cortex which are thickened, in which considerable numbers of large lymphoid cells are found; another node (148) reveals a thinner diffuse cortical tissue, but one in which the cortical sinuses are in certain areas full

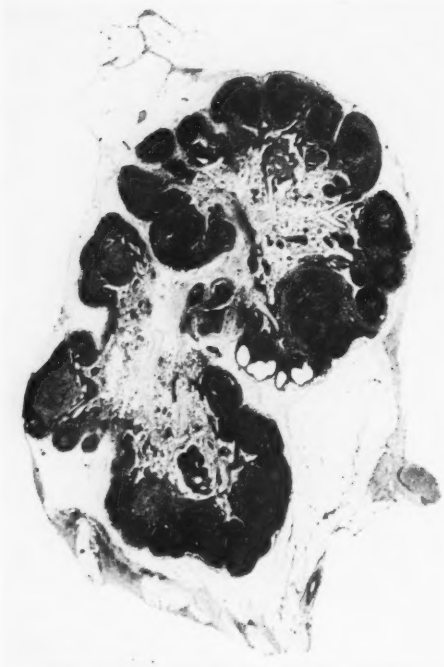


FIG. 4

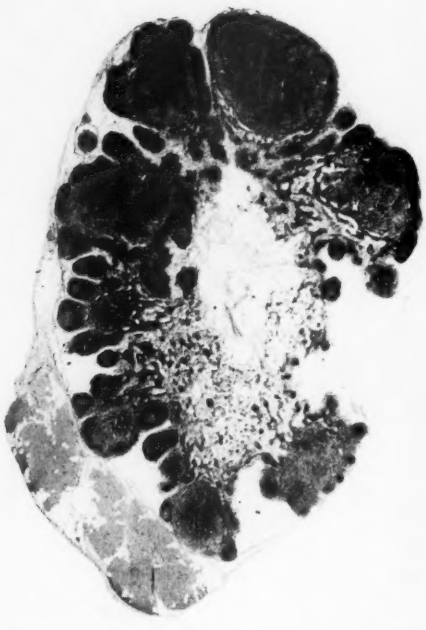


FIG. 5

Figure 4—Control lymph node showing germinal centres, thin diffuse cortical tissue and poorly developed medullary trabeculae. (×15.) Rabbit 82. Figure 5—Control lymph node showing germinal centres, moderately thick diffuse cortical tissue and poorly developed medullary trabeculae. (×15.) Rabbit 66.

of small lymphocytes; a third node (160) reveals a cortical pattern similar to that of the control nodes. The new formation of germinal centres in the reactive or previously reactive sectors of the cortex is seen in all the ten-day nodes. These new centres are small, capped with a peripheral collection of small lymphocytes, are usually bare towards the medulla and contain a variable number of pyroninophilic cells, many of which are of large lymphoid cell type. In nodes removed twelve days after grafting the newly formed germinal centres are again evident, diffuse cortical tissue is thinner, and the content of large lymphoid cells is low. After fourteen days it is difficult to decide which parts of the diffuse cortical tissue have been reactive, but the finding of newly formed germinal centres, or the presence of numerous small lymphocytes in the cortical sinuses, is probably indicative that the cortex bearing these changes is resolving. In the node removed twenty-one days after grafting a histological appearance similar to that of the control nodes is seen.

The medullary trabeculae of the four-, five-, six- and eight-day nodes show an enlargement which is due to an increased number of small lymphocytes, large lymphoid cells and mature plasma cells. By ten days germinal centres are apparent in the medulla, and the trabeculae appear thinner, a feature which is also seen in the twelve- and fourteen-day nodes. In the node removed three weeks after grafting a normal appearance is present.

In the sinuses of the medulla macrophages packed with ingested erythrocytes are found in the two-day nodes (Fig. 11) and large lymphoid cells appear in great numbers in the four-, five-, six- and eight-day nodes (Fig. 12). After ten days one node (148) contains a large number of small lymphocytes and a few large lymphoid cells in the cortical sinuses, an appearance which is seen in lesser degree in some of the twelve- and fourteen-day nodes. The cellula

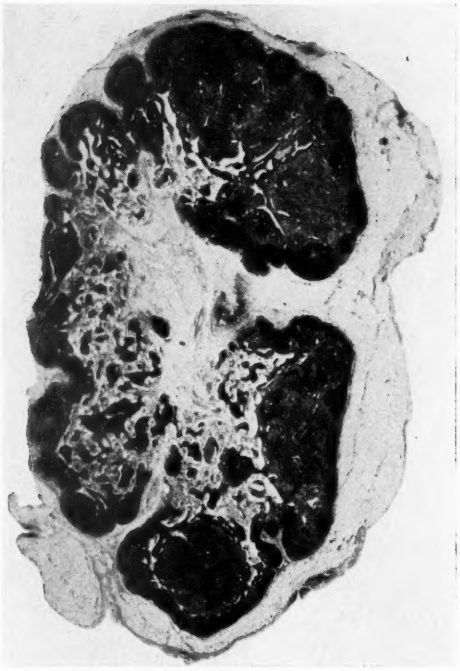


FIG. 6

Lymph node removed six days after the transplantation of homologous cancellous bone into its drainage area. The diffuse cortical tissue is greatly thickened especially towards the upper pole of the node. Germinal centres are present. The medullary trabeculae are hyperplastic. (×15.) Rabbit 82.

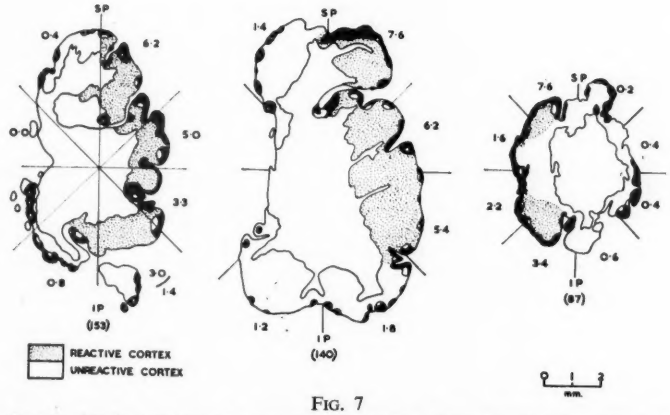


FIG. 7

Camera lucida tracings of a section from each of three lymph nodes removed four days after insertions of homografts of cancellous bone into their drainage areas. The stippled areas indicate the sectoral distribution of the large and medium lymphoid cells. See text for an explanation of the numerals situated at the periphery of each section. (SP=superior pole; IP=inferior pole.)

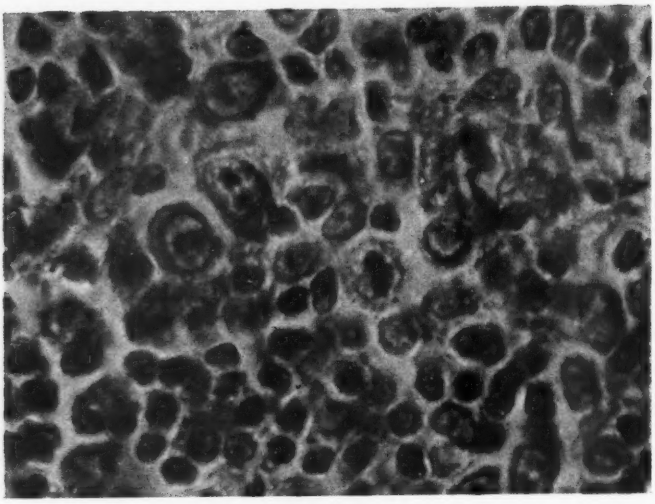


FIG. 8

Large and medium lymphoid cells in the reactive diffuse cortical tissue of a lymph node removed five days after the transplantation of homologous bone into its drainage area. The cytoplasm is pyroninophilic, the chromatin is pale and the nucleoli are stained red. A few small lymphocytes are seen. Oil immersion. (×980.) Methyl green-pyronin. Ektachrome. Rabbit 142.

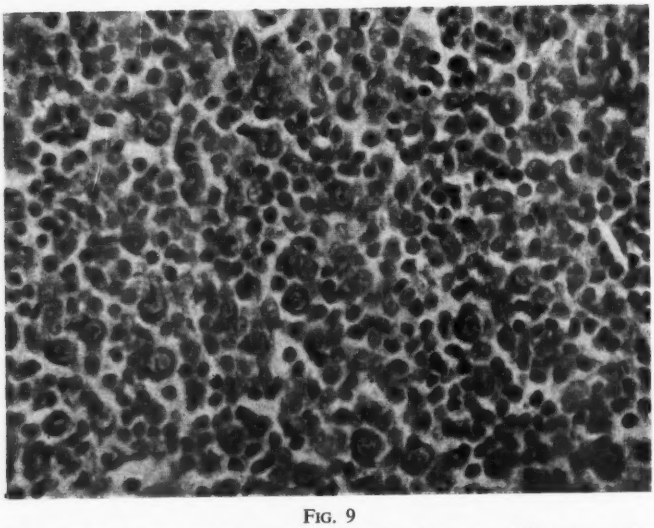


FIG. 9

The reactive diffuse cortical tissue of a lymph node removed four days after the transplantation of homologous bone into its drainage area. Distributed amongst the small lymphocytes are a considerable number of large and medium lymphoid cells which have red (pyroninophilic) cytoplasm and nucleoli. (×420.) Methyl green-pyronin. Ektachrome. Rabbit 140.

content of the medullary sinuses three weeks after insertion of the graft is not different from the control nodes.

Sectoral distribution of the large lymphoid cell response—The distribution of the reactive and unreactive parts of the cortex in three nodes from three homografted animals is shown in Figure 7. The sectoral pattern of the large lymphoid cell response is clearly shown. To aid the establishment of this observation, counts of large and medium lymphoid cells have been made in the eight sectors produced by the division of one histological section from each of the twelve nodes, by four lines as shown in Figure 7 (Serial No. 153). In each section counts

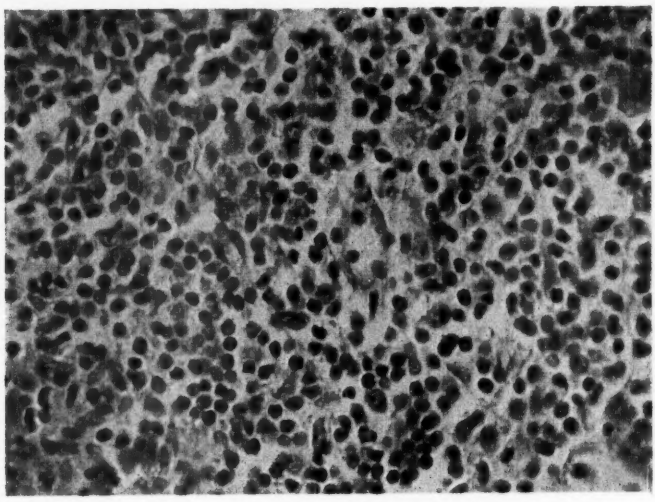


FIG. 10

The unreactive diffuse cortical tissue of the same lymph node as that shown in Figure 9. Small lymphocytes are the main type of cell present, and large and medium lymphoid cells are virtually absent. (×420.) Methyl green-pyronin. Ektachrome.

were made where possible in five separate fields under oil immersion (eyepieces, ×10; objective, ×100), each field representing an area 8×8 divisions of the graticule. The higher concentration of the large lymphoid cells in the reactive parts of the diffuse cortical tissue, indicated by the figures in each sector, is apparent.

Quantitative analysis of the large lymphoid cell response—Counts of the numbers of large and medium lymphoid cells in the diffuse tissue of the cortex of three control nodes are shown in Table III.

In the control nodes it will be seen that very few large lymphoid cells are present in the diffuse tissue of the cortex.

In animals receiving homografts, counts made in the reactive parts of the diffuse lymphoid tissue of the cortex are shown in Table IV together with a statistical analysis of the data.

Though the reactive cortex was easily discernible in the four-, five-, six- and eight-day nodes, the reactive cortex was more difficult to assess in the two-, ten-, twelve- and fourteen-day nodes. However, some indication of reactivity could often be achieved by the recognition of large lymphoid cells in the diffuse cortical tissue, collections of newly formed germinal centres or the presence of masses of small lymphocytes in the deeper sinuses of the cortex.

The mean number of large and medium lymphoid cells per unit area of reactive diffuse cortical tissue in three animals at each time interval after the insertion of the homograft is shown in Figure 13 together with the standard error for each day studied after operation.

TABLE III

COUNTS OF LARGE AND MEDIUM LYMPHOID CELLS IN THE DIFFUSE CORTICAL TISSUE

Control lymph nodes

Serial number	Large lymphoid cells		Standard error
	Counts per unit area	Mean number and standard deviation	
98	0·56		
106	0·61	0·57±0·04	0·02
112	0·54		

TABLE IV

COUNTS OF LARGE AND MEDIUM LYMPHOID CELLS IN THE DIFFUSE CORTICAL TISSUE

Homograft lymph nodes

Serial number	Days after grafting	Large lymphoid cells		Standard error	P. Homograft/ control
		Counts per unit area of reactive cortex	Mean number and standard deviation		
86	2	0·63			
162	2	0·74	0·74±0·11	0·06	0·05<P<0·1
178	2	0·85			
87	4	3·90			
140	4	3·91	4·48±1·00	0·58	0·001<P<0·01
153	4	5·63			
141	5	6·38			
154	5	6·99	6·47±0·48	0·27	<0·001
185	5	6·04			
89	6	3·15			
134	6	2·76	3·84±1·55	0·89	0·02<P<0·05
142	6	5·61			
90	8	4·06			
143	8	3·58	3·76±0·08	0·04	<0·001
155	8	3·65			
148	10	1·27			
149	10	1·60	1·43±0·05	0·03	<0·001
160	10	1·42			
93	12	0·27			
150	12	0·72	0·85±0·66	0·38	0·4<P<0·5
161	12	1·57			
112	14	0·69			
151	14	0·44	0·67±0·07	0·04	0·05<P<0·1
159	14	0·88			

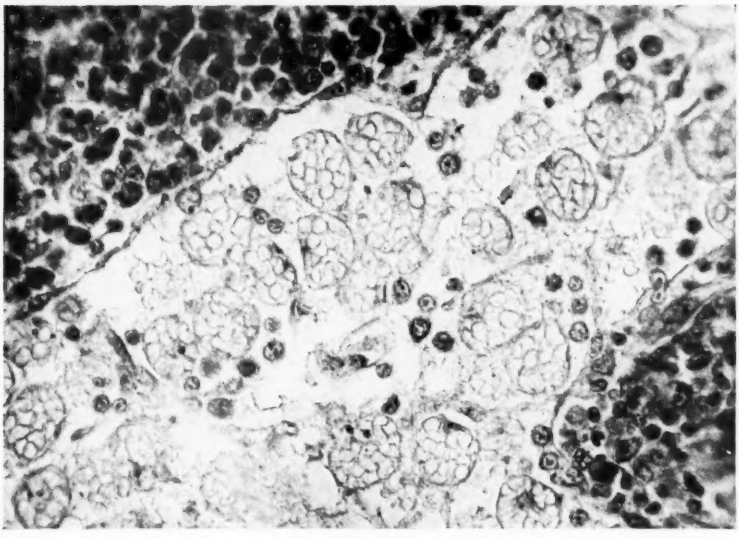


FIG. 11

Medullary sinusoid of a lymph node removed two days after the transplantation of homologous cancellous bone into its drainage area. The sinusoid contains large numbers of macrophages with ingested erythrocytes, small lymphocytes and a few pyroninophilic cells. (×280.) Methyl green-pyronin. Rabbit 162.

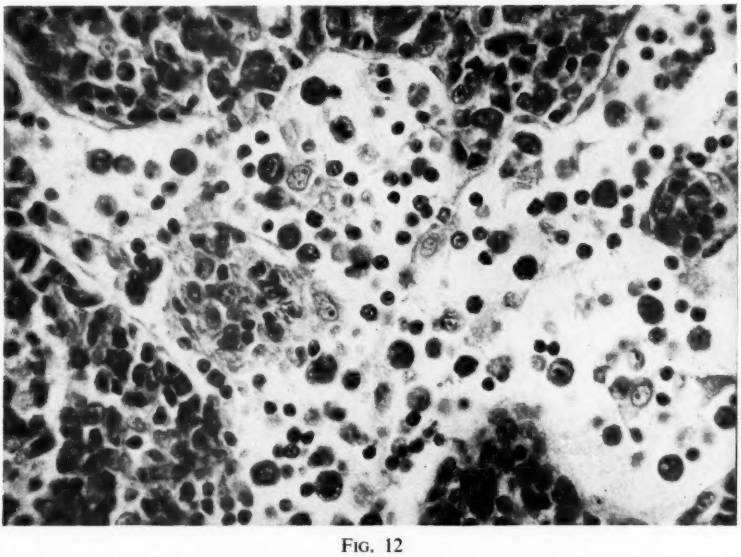


FIG. 12

Medullary sinusoid of a lymph node removed six days after the transplantation of homologous cancellous bone into its drainage area. The sinusoid contains large and medium lymphoid cells, small lymphocytes and a few macrophages which do not contain phagocytosed erythrocytes. These cells in this sinusoid would have gained the general circulation. (×280.) Methyl green-pyronin. Rabbit 142.

The mean maximal response of the large lymphoid cells is achieved at the fifth day after grafting. The large lymphoid cellular response of the autografted nodes has not been assessed quantitatively, because it is felt that to distinguish cortex which has been exposed from cortex which has not been exposed to the lymphatic drainage of the graft is not possible. Any count therefore in autografted nodes would include all the diffuse tissue of the cortex, and hence comparison with counts from the homografted nodes would not be possible.

Second regional nodes—There does appear to be an increase in the diffuse lymphoid tissue in certain of these nodes, but with occasional exceptions it is not accompanied by an increase in the concentration of large lymphoid cells. The medullary trabeculae show little hypertrophy and the sinuses in the four-day nodes contain numerous macrophages, but at later stages large lymphoid cells and small lymphocytes predominate.

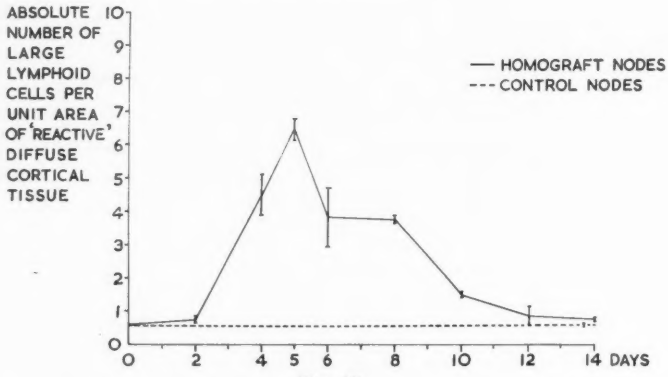


FIG. 13

Graph of the mean count (with standard errors) of the large and medium lymphoid cells per unit area in the reactive diffuse cortical lymphoid tissue of nodes draining homografts of cancellous bone inserted into their drainage areas at intervals up to fourteen days previously. The interrupted line represents the mean count per unit area of the large and medium lymphoid cells in the diffuse cortical lymphoid tissue of three control nodes.

Autograft nodes—The histological appearances of the first regional nodes from the autografted ear are only slightly different from the control nodes. The diffuse cortical tissue appears to be slightly thickened in the six- (Fig. 14) and eight-day nodes, but without any apparent increase in the large lymphoid cell concentration (Figs. 15 and 16). The medullary trabeculae are thickest in those nodes removed six to eight days after autografting. In the medullary sinuses macrophages containing erythrocytes are to be seen on the fourth day, and large lymphoid cells on the eighth day after operation, but neither of these two cell types occurs in the profusion seen in the medullary sinuses of the homografted nodes. By the tenth day the nodes have a normal histological appearance, though occasional germinal centres are seen in the medulla.

The second regional nodes reveal a distinct thickening of the diffuse cortical tissue, in which the large lymphoid cell content remains low. The medullary trabeculae show little change and the cell content of the medullary sinusoids is similar to that of the control nodes, apart from an increase of macrophages in the early stages.

Lungs, Mediastinal Nodes and Spleens

Careful comparison of the lungs and mediastinal lymph nodes of the homografted and autografted animals fails to reveal any difference in histological appearance between the two series.

Histological examination of the lymphoid tissue of the spleens from the homografted and the autografted animals does not show any difference in the appearance of the Malpighian corpuscles, red pulp or the sinusoids. There does appear, however, to be more haemosiderin contained in macrophages in the homograft spleens removed on the fourth to the eighth days after operation than in any of the other spleens examined.

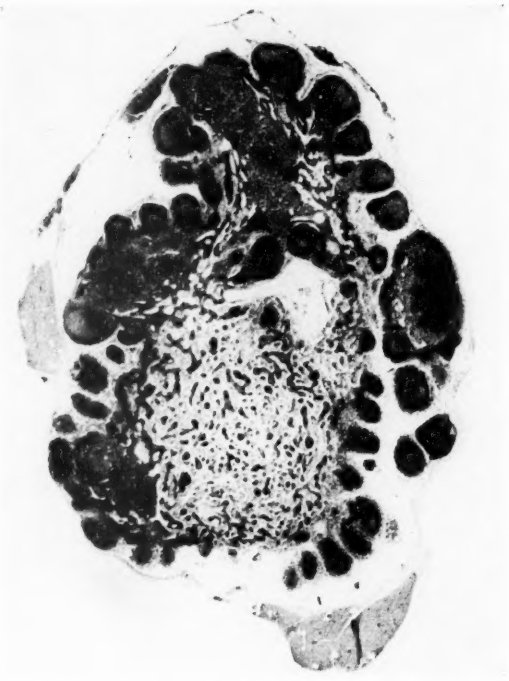


FIG. 14
Lymph node removed six days after the transplantation of autologous cancellous bone into its drainage area. The diffuse cortical tissue is moderately thickened and germinal centres are present. The medullary trabeculae are moderately hyperplastic. (×15.) Rabbit 66.

Bone Grafts

The autografts—Some osteocytes, judged by the staining appearances of their nuclei, appear to survive in all specimens, particularly in the remaining cortex and certain trabeculae of the graft. The longer the time interval after insertion of the implant the fewer are the number of living osteocytes. At three weeks a few osteocytes are still viable. The first new bone is seen in grafts removed six days after operation; later, new bone appears in association with peripherally situated trabeculae. This new formation of bone is present in all grafts removed on and after the eighth day, and continues through the stages studied until nearly all the peripheral bone trabeculae have a covering of new bone. The bone marrow appears necrotic and invasion by capillaries, round cells, fibroblasts and polymorphs is bringing about replacement. Fat cells are removed more slowly than the myeloid elements. The surrounding tissue is mainly composed of fibroblasts, small round cells and a few polymorphs.

The homografts—Certain cortical and trabecular osteocytes appear to survive in all specimens. The number surviving decreases with the length of time after operation. A few osteocytes are

still living in the remaining cortex of the graft removed three weeks after the operation. The first new bone is seen in a graft removed five days after operation. In most of the grafts removed on and after the *sixth* day a small amount of new bone is present, either subperiosteally or in association with peripheral trabeculae. The covering osteoblastic cells of the trabeculae are flattened and the staining qualities of their nuclei are abnormal. The quantity of this dead new bone is small in all the homografts studied. Cellular invasion of the homografts, more marked than in the autografts, is first apparent six to eight days after operation and is seen especially in relation to the subperiosteal surface. The cells consist mainly of small lymphocytes, though eosinophils or polymorphs occur to a lesser degree; large lymphoid cells are not seen in any of the specimens examined. The bone marrow is necrotic and replacement is taking place by a vascular and cellular invasion.

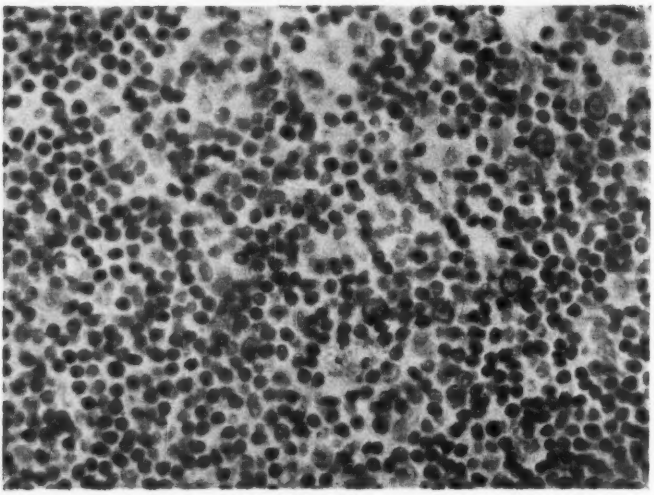


FIG. 15

The diffuse cortical tissue of a lymph node removed four days after the transplantation of autologous bone into its drainage area. A few large and medium lymphoid cells are present amongst the small lymphocytes. (×420.) Methyl green-pyronin. Ektachrome. Rabbit 98.

DISCUSSION

The findings described above are consistent with the view that the immune reaction to a primary homograft of fresh cancellous bone inserted into the subcutaneous tissues takes place mainly in the first regional node draining the graft. This conclusion agrees with knowledge obtained by using skin grafts, where the principal immunological response also occurs in the regional nodes draining the grafts (Billingham and colleagues 1954, Scothorne and McGregor 1955), though it must be remembered that the homograft reaction is systemic (Medawar 1945).

Serological investigations after the insertion of homografts of bone, or the injection of homologous bone extracts, have in many instances failed to demonstrate the presence of humoral antibodies (Burwell and Gowland 1961). It seems that the immunological response to fresh homografts of bone will prove to be similar to the response which has been more fully investigated using skin homografts where the immune state is due to " endoantibodies " (Medawar 1958) carried by cells of the lymphoid series. Thus it has been shown that passive immunity to a skin homograft may be transferred by a suspension of cells from the regional nodes of the operated side, not at all by non-regional nodes and only to a small extent by cell suspensions from the spleen (Billingham and colleagues 1954).

A histological study of the reaction occurring in the regional node draining skin homografts was first made by Gallone and colleagues (1952), who found an increase in the pyronin-staining elements in the nodes. Scothorne and McGregor (1955) confirmed these observations and introduced the term "large lymphoid cell" to describe the cell type which appeared in great numbers in the cortex, and to a lesser extent in the medulla, of lymph nodes draining skin homografts. The large lymphoid cells they described as ". . . round or ovoid in outline, about 15–17μ in greatest diameter, and have large pale nuclei, one or more prominent nucleoli and strongly pyroninophilic, rather vacuolated, cytoplasm." Our observations confirm a similar production of "large," but also of "medium-sized, pyroninophilic lymphoid cells" occurring in the first regional nodes draining homografts of fresh cancellous bone.

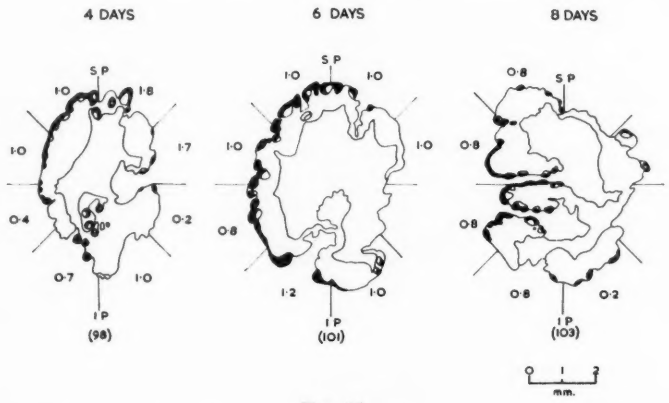


FIG. 16

Camera lucida tracings of a section from each of three lymph nodes removed four, six and eight days respectively after the insertion of autografts of cancellous bone into their drainage areas. For further details see Figure 7.

Scothorne (1957) has drawn attention to the resemblances of the large lymphoid cell of the homograft response to the cells that are usually regarded as antibody producers. It is probable that the large lymphoid cells appearing in a lymph node draining the site of a recently inserted homograft belong to the same general family of cells that produce humoral antibodies against bacteria, vaccines and other soluble antigens (Oakley 1959). Recently a fluorescent technique has shown the presence of a high concentration of antibody in the large cells which develop in lymph nodes responding to soluble antigens (Leduc, Coons and Connolly 1955).

The Cell Reaction to Antigens

Nomenclature—Various names have been applied to the large and medium sized cells with pale nuclei, prominent nucleoli and intensely basophilic or pyroninophilic cytoplasm appearing in lymph nodes subjected to a wide variety of antigenic challenges. The names which have been used include: proliferating endothelial (reticular) cell (Marchand 1924), free stem cell or haemocytoblast (Leduc, Coons and Connolly 1953; Fagraeus 1960), blast cell (Leduc and her colleagues 1953, 1955), lymphoblast or acute splenic tumour cell (Rich, Lewis and Wintrobe 1939), large lymphocyte (Shepro, Eidelhoch and Patt 1960), reticular lymphocyte (Sundberg 1955), young lymphocyte (Harris and Harris 1949), lymphogonia (Unno, Hanaoka, Iwai, Hashimoto and Morita 1954), transitional cell (Fagraeus 1948; Radici and Piredda 1957), immature plasma cell (Fagraeus 1948), activated reticulum cell and plasmablast (Marshall and White 1950), and reticular plasma cell (Kolouch, Good and Campbell 1947). All these terms are unsatisfactory, either because they are insufficiently comprehensive (acute splenic tumour

cell), or because they unjustifiably assume a knowledge of the origin (proliferating endothelial cell, activated reticulum cell, reticular lymphocyte, reticular plasma cell, transitional cell), or of the fate (immature plasma cell, plasmablast, lymphoblast, large lymphocyte, young lymphocyte, lymphogonia) of the cell. Though the evidence suggests that large and medium lymphoid cells may be the progenitors both of mature plasma cells and of small lymphocytes (see below), we prefer the use of a more non-committal term. But so as to connote the known association of antibody production with cells of the lymphoid series, we agree with the term " large lymphoid cell " proposed by Scothorne and McGregor. In order to include cells of a similar morphology but smaller size we would describe the smaller cell as a " medium lymphoid cell," as indeed Craigmyle has done. The use then of the terms free stem cell, haemocytoblast or blast cell are unsatisfactory. However, Harris (1960) has stated ". . . it would be wise to reserve as much as possible any implication of finality in the cytological nomenclature that we adopt in relation to the synthesis of antibody . . ."

The stimulus for cellular response—The stimulus for the production of the large and medium lymphoid cells is most probably provided by the iso-antigens (Medawar 1959*a* and *b*) in the cells of the homograft. It is not known whether the relatively stable antigens capable of causing the production of humoral antibodies (" H-antigens ") or the unstable antigens provoking transplantation immunity (" T-antigens ") are the stimulus to the formation of " activated " lymphoid cells. It is possible that both groups of iso-antigens play roles in the large lymphoid cell response to a homograft. It must be remembered that the cell population of a cancellous bone homograft is complex. Further work (Burwell 1960*b*) has revealed that the main antigenic component of our homografts is the nucleated cells of the red bone marrow, though under certain conditions newly formed homograft bone is apparently antigenic.

The origin of the lymphoid cells—The source of the large and medium lymphoid cells is not clear. Fagraeus (1948) produced evidence that reticulo-endothelial cells were the stem cells of antibody production. Marshall and White (1950) favoured the undifferentiated mesenchymal cells (primitive reticular cells) as the precursors, and this view has now received wide support (Fagraeus 1960). However, Holub (1960) showed that thoracic duct lymph, placed with a heterologous antigen in a diffusion chamber which was then implanted intraperitoneally in newborn rabbits, would not only produce antibody, but also pyroninophilic cells. Roberts, Dixon and Weigle (1957) showed that cell suspensions containing both macrophages and lymphocytes injected subcutaneously or intramuscularly into x-irradiated immunologically inert rabbits resulted in the transformation of these cells, after antigenic challenge, into large pyroninophilic antibody-producing cells. However, Terasaki (1959) has shown that in the peripheral blood of chickens, it is the lymphocytes and not the monocytes which are the immunologically competent cells. Some light may have been shed upon the early phases of antibody production by recent observations (Sharp and Burwell 1960*a*), which have shown an interaction between macrophages and lymphocytes (" peripolesis ") in lymphoid tissue obtained from antigenically challenged animals.

The fate of the lymphoid cells—The fate of the large and medium lymphoid cells is still unsolved. In our material there is histological evidence to show that at least some of the large and medium lymphoid cells do gain access to the venous circulation, for numerous cells of this type are found in the medullary sinuses in nodes removed four, five, six and eight days after insertion of the bone grafts. No histological evidence can be found to show that the large cells are filtered out in the pulmonary capillary bed, as occurs with malignant cells (Willis 1950) and also under certain conditions with macrophages (Easton 1952). It seems probable however that the large and medium pyroninophilic cells metastasise to lymphoid tissue at other sites in the body and there by mitosis produce generations of immune cells. An increased number of cells of large lymphoid type has been detected, after challenge with soluble antigens, in the lymph coming from lymph nodes (Ehrich and Harris 1942; Amano, Unno, Hanaoka and

Tamaki 1951) and in the peripheral blood (Unno and colleagues 1954). Some workers have suggested that the large lymphoid cell (or its equivalent) gives rise to small lymphocytes (Ehrich 1929; Rich and colleagues 1939; Harris and Harris 1949, Unno and colleagues 1954). It has been shown more recently that large lymphoid cells can develop into mature plasma cells (Fagraeus 1948; Marshall and White 1950; Ringertz and Adamson 1950; Keuning and Van der Slikke 1950; Leduc and colleagues 1953). Indeed, Scothorne (1957) has suggested that the mature plasma cell may develop only in response to a particular intensity and duration of antigen stimulus. It seems possible therefore that under different antigenic conditions the large lymphoid cell may develop into either a mature plasma cell or a small lymphocyte. On the other hand Thiéry (1960) and Bernhard and Granboulan (1960) maintain that the lymphocyte series and the plasma cell series are immutable. But Harris (1960) has pointed out that morphological differences between cells as revealed by the light and electron microscopes may have less significance when one appreciates the very rapid rate at which dynamic changes occur in cells.

The importance of immune cells—The importance of immune cells in the destruction of tissue homografts has been convincingly demonstrated by the use of membranes impermeable to cells (Algire, Weaver and Prehn 1954; Weaver, Algire and Prehn 1955; Woodruff 1957). It is not definitely known which type of immune cell is the one which exerts the destructive effect upon a homograft (Darcy 1952). It is unlikely that the large and medium lymphoid cells themselves play any direct role in the rejection of primary homografts either of skin (Scothorne 1957), or of bone, because these large cells are absent from the bed of our homografts. It seems more probable that small lymphocytes produced by mitosis from these large lymphoid cells are the responsible cellular agents of graft rejection. The small lymphocytes first become evident in large numbers in the bed of our homografts about the sixth day after operation and their appearance is associated in time with the cessation of the formation of new bone. It is still not clear how the immune cells destroy normal tissue homografts (Berrian and Brent 1958).

The sectoral distribution of the large and medium lymphoid cells throughout the diffuse cortical lymphoid tissue in the homograft nodes of the writers' material is probably determined by the entry of the iso-antigens into that particular part of the cortex. To test this probability 0·1 millilitre of indian ink was injected subcutaneously on the ventral aspect of a rabbit's ear one centimetre from the external auditory meatus. The first regional node was removed half an hour later, fixed, embedded and sectioned. It was seen that the indian ink was distributed only to one side of the node. Hence it is concluded that the localised entry of the antigens through certain lymphatics to the first regional node determines the sectoral distribution of the large and medium lymphoid cell response. It is not definitely known whether the antigenic material from the homograft reaches the first regional node contained within cells or in the extracellular fluid of the afferent lymph. However, the experiments of Woodruff (1957) which showed in rats that split-skin homografts protected from contact with host cells by a millipore membrane impermeable to cells were not effectively antigenic, suggest that cells either from the host or the graft are involved in the transport of the iso-antigens to the regional node. The recognition of a sectoral distribution of reactive cells within the regional node draining a tissue homograft is important in the interpretation of experiments designed to demonstrate the homograft reaction *in vitro*, in which small explants of the reactive regional node are set up against the donor tissue (Scothorne and Nagy 1960; Sharp and Burwell 1960*b*).

Other workers have approached the quantitative analysis of lymph nodes in different ways. Sjövall and Sjövall (1930) analysed the proportions of cortex, secondary nodules and medulla in lymph nodes recently exposed to pseudomonas pyocyanea infection. Carlson and Gyllensten (1958) studied plasma cells in the growing lymphatic system of young guinea pigs. They counted 500 cells " at random " in both cortex (excluding germinal centres) and medulla. Ernström and Gyllensten (1959) have applied the same technique to lymphatic hyperplasia

caused by thyroxine administration. But it is difficult to apply the same random sampling of cells in the cortex or medulla of nodes which are reacting to an antigenic challenge, owing to the marked differences in the distribution of the cellular responses in different parts of the same node. However, Radici and Piredda (1957), who studied the cellular response in lymph nodes reacting to skin homografts, analysed the proportions of different cell types present in the medullary trabeculae at three, five, seven, nine and eleven days after homografting. They found a rapid rise in the number of " transitional cells," which reached a maximum of 7·3 per cent on the seventh post-operative day, but on the ninth day mature plasma cells had become more numerous than transitional forms. Craigmyle (1958) investigated the reaction of lymph nodes to homografts and heterografts of costal cartilage and commented upon the difficulty of attempting a quantitative analysis of nodes in which the distribution of reactive cells was so heterogenous. Shepro and colleagues (1960) have described the lymph node responses to malignant homografts and heterografts in the hamster. In the cortex of each node they counted the number of secondary centres and measured the ratio of large to small lymphocytes in ten areas lying close to and between the secondary centres. They also measured the percentages of various cell types in the medullary portions of the nodes. Nossal (1959) investigated the lymph node response in rabbits to a bacterial antigen injected locally and attempted to overcome the difficulty of uneven cellular distribution of immature plasma cells by using smears of the regional node and performing a differential cellular count on 500 to 1,000 cells. The writers, like Scothorne (unpublished), attempted to analyse quantitatively the reactivity of lymph nodes to homografts by means of smears, but abandoned it in favour of a direct analysis of the most reactive part of the node, namely the diffuse lymphoid tissue of the cortex. It has been shown that a variable amount of the diffuse cortical lymphoid tissue does not react. It is considered justifiable to ignore this unreactive cortex in the quantitative analysis of the large and medium lymphoid cell responses in the homografted nodes. There is a marked irregularity in the distribution of large and medium lymphoid cells in the reactive part of the diffuse lymphoid tissue of the cortex. Thus, frequently the upper pole of the node reveals a higher concentration of these cells than do the more inferior parts of the reactive cortex. Again, usually there are more large and medium lymphoid cells in the outer than in the juxta-medullary parts of the cortex, and occasionally the same sector of cortex reveals a different number of pyroninophilic cells in the three different histological sections studied for the quantitative analysis of each node. Hence the variation of the distribution of large and medium lymphoid cells is three-dimensional. The counting of cells in the reactive cortex at short distances along " isobaths " at different distances from the capsule in three different sections from the same node is an attempt to produce a reasonable assessment of the large and medium lymphoid cell responses occurring in the node.

The time sequences of the large lymphoid cell response in the first regional lymph nodes draining our bone homografts are in agreement with those reported by Mitchison (1955) who found that the competence of regional nodes to transfer immunity caused by a regional tumour homograft in mice, began three to five days and ended ten to fifteen days after implantation. However, Billingham and colleagues (1954) using skin homografts in mice, were able to show that the regional nodes retained some capacity to transfer adoptive acquired immunity when removed thirty days after skin transplantation.

Changes in the graft—It is now well established that a few osteocytes survive in autografts of cancellous bone (Gordon and Ham 1950; Hutchison 1952; Chalmers 1959). Our finding that some osteocytes survive in both the cortex and certain trabeculae of iliac homografts agrees with the observations of Hutchison (1952) and Chalmers (1959), though Ham and Harris (1956) maintain that homologous osteocytes are destroyed. More recently, however, Heslop, Zeiss and Nisbet (1960) have reported the continued survival of osteocytes in homologous cortical tibiae implanted into subcutaneous sites in rats. These workers, like Chalmers (1959), attribute the continued survival of the osteocytes to an inability of host inflammatory cells to

gain access to the bone cells through the canaliculi. Craigmyle (1958) explains the prolonged survival of cartilage homografts by a protective effect of the matrix on the cartilage cells. Heslop and her colleagues also found that the longer the time after insertion of a bone homograft in the rat, the fewer were the surviving osteocytes, an observation which we confirm in rabbit cancellous bone. Further, Heslop and her colleagues stated that after thirty-five days significantly more osteocytes survived in their autografts than in their homografts, which they interpreted as being due to changes in local vascularity.

No new bone is seen in our homograft material before the fifth day, though in rats Chalmers (1959) found new bone four days after bone homografting. The quantity of new bone formed by the homografts in the writers' material is much less than in the autografts, and at the time when the cellular infiltration of the graft bed is first apparent (six to eight days), the staining appearances of the covering osteoblasts of the newly formed bone become abnormal. It is tempting then, to correlate the mean maximal large lymphoid cell response at five days (with its probable attendant production of immune small lymphocytes) with the first appearance of cellular infiltration of the graft bed at six to eight days. It is likely that this cellular infiltration of bone homografts is related to the cessation of further new bone formation. However, Heslop and her colleagues (1960) maintain that osteogenesis is not specifically inhibited by the inflammatory cells, but is impaired by an abnormality in the connective tissue developing around the homografts. They interpret the abnormal connective tissue as being ". . . a direct outcome of the presence of the homologous bone . . .," that is due to a histo-incompatibility. The suggestion that the first phase of new bone formation in homografts is impaired by an abnormal connective tissue is untenable for these reasons: 1) New bone ceases to be produced in a homograft about the time of the maximal lymphocytic infiltration of the graft (Chalmers 1959), an observation which we confirm. 2) The importance of immune cells of the lymphoid series in the destruction of both tumour and skin homografts has been convincingly demonstrated experimentally (Mitchison 1954; Billingham and colleagues 1954). It is unlikely that bone which has antigens in common with skin (Chalmers 1959) would be rejected by a different mechanism. 3) No new bone occurs either early or late around homografts of bone in rats which have received skin from the same donors three weeks previously (Chalmers 1959). These experiments suggest that the early phase of new bone formation in bone homografts is due to the survival and proliferation of the grafted cells which are then destroyed by the immune responses of the host. Chalmers also described a *second* phase of new bone formation in some fresh bone homografts four or more weeks after implantation. Some freeze-dried bone homografts, in which it was thought all the grafted cells had been killed, also showed small amounts of new bone characteristic of the late phase of homograft new bone formation. Chalmers attributed this later formation of new bone to metaplasia of the connective tissues of the host. It is possible then that an abnormal connective tissue would prevent a late phase of bone formation. It is unlikely that the abnormal connective tissue which Heslop and her colleagues describe as developing around their bone homografts is a direct outcome of the presence of the homologous bone, because tissues from different animals of the same species cultured *in vitro* will grow towards each other, join and may even grow into each other without any evidence of an adverse effect of the tissue from one animal upon the tissue of the other (Harris 1943; Medawar 1948; Grobstein and Youngner 1949; Laviolette and Rerolle 1958; Scothorne and Nagy 1960). It is more likely that the disturbances of fibroblasts and collagenous fibres which Heslop and her colleagues described in relation to their homografts result from an effect of immune cells either directly upon the local tissues or indirectly upon the local capillary walls. Indeed Heslop and her colleagues suggested that the hyaline change which they observed in the local capillary walls may have resulted from prolonged stimulation of the antibody-forming system.

Chalmers (1959) recorded that in rats, when homologous cancellous bone is transplanted to a muscular site a large amount of new bone may occasionally be produced. The dissolution

of this bone frequently does not start until sixteen or even eighteen days after operation. Moreover, Chalmers has shown that both phases of new bone formation around bone homografts are prevented if the animals have received skin homografts from the same donors three weeks previously; but if the initial challenges are not skin homografts transplanted on to subcutaneous sites, but are bone homografts transplanted into skeletal muscles, then there is a less consistent suppression of the early and late phases of new bone formation. From this experiment Chalmers concluded that muscle compared with skin is a less satisfactory implantation site from which the immunity may be evoked. This difference is probably due to the fewer lymphatic vessels in skeletal muscle, compared with skin, allowing a smaller amount of the iso-antigens to reach the regional lymph nodes from homografts implanted into muscle than from those implanted into the subcutaneous tissues. Hence a higher proportion of iso-antigens will enter the blood stream directly from muscular than from subcutaneous sites; the dilution of the iso-antigens by the blood thus may weaken the stimulus to the production of immune cells.

CONCLUSION

It is clear that measurement of the weight changes occurring in the first regional lymph nodes draining areas which have recently received homografts of fresh bone, and quantitative assessment of the occurrence of large and medium lymphoid cells as a histological indicator of the immune response developing in the node, provide a method for comparison of the antigenicity of the components of fresh bone and also the antigenicity of different types of stored bone. This aspect will be considered in a later communication.

SUMMARY

1. The effects of the insertion of pieces of fresh cancellous bone into the subcutaneous tissues of the ear upon lymph nodes and spleens have been investigated in seventy rabbits.

2. The main immunological response is found to occur in the first regional nodes draining the sites of insertion of homografts of bone, which show a considerable increase in weight compared with nodes draining autografts of bone.

3. An increased number of large and medium lymphoid cells occurs principally in the first regional node of the homografted animals, as Scothorne and McGregor (1955) observed using skin as the homografted tissue.

4. The large and medium lymphoid cell response is found in both the cortex and the medulla of the lymph nodes. In the cortex a sectoral distribution of the cellular response is observed and the name reactive cortex is given to these sectors. Evidence is presented to show that the sectoral pattern of reactivity is probably determined by the localised entry into the node of iso-antigens through lymphatic vessels draining the bed of the graft.

5. We have made a quantitative analysis of the large and medium lymphoid cell response in the reactive parts of the diffuse lymphoid tissue of the cortex. The mean maximal large and medium lymphoid cell response occurs five days after the insertion of bone homografts.

6. The origin and fate of the large and medium lymphoid cells and their role in the production of antibodies is reviewed in the light of recent work.

7. A correlation is made between the maximal production of large and medium lymphoid cells in the first regional lymph node, the invasion of the graft bed with small lymphocytes and the inhibition of new bone formation in the homografts.

The writers are indebted to Professor A. Durward for facilities to carry out the present work and for his continual encouragement. We also wish to acknowledge the encouragement of Mr A. B. Pain and Mr J. M. P. Clark in whose department this series of studies on bone immunology commenced when one of the writers (R. G. B.) was their Registrar. Mr Clark gave valuable aid with translations. Professor P. B. Medawar, F.R.S., and Professor R. J. Scothorne very kindly read the manuscript and made valuable suggestions. We wish to extend our grateful thanks to Dr J. A. Holgate, Assistant Director of the Department of Pharmacological Research, Parke Davis & Co., and also to Dr B. T. Warner, Biometrician to Parke Davis & Co., for their statistical survey of the data. Also we wish to thank Miss Mary Brown for Figure 1, and Mr R. K. Adkin, A.I.S.T., and Mr R. H. Nettleton for technical assistance.

REFERENCES

ALGIRE, G. H., WEAVER, J. M., and PREHN, R. T. (1954): Growth of Cells *in vivo* in Diffusion Chambers. I. Survival of Homografts in Immunized Mice. *Journal of the National Cancer Institute*, **15,** 493.

AMANO, S., UNNO, G., HANAOKA, M., and TAMAKI, Y. (1951): Studies on the Discrimination of Lymphocytes and Plasma Cells. Supplements on Advocation of the "Lymphogonia" Theory. *Acta Pathologica Japonica*, **1,** 117.

BERNHARD, W., and GRANBOULAN, N. (1960): Ultrastructure of Immunologically Competent Cells. In *Ciba Foundation Symposium on Cellular Aspects of Immunity*. London: J. & A. Churchill Ltd.

BERRIAN, J. H., and BRENT, L. (1958): Cell-bound Antibodies in Transplantation Immunity. *Annals of the New York Academy of Sciences*, **73,** 654.

BILLINGHAM, R. E., BRENT, L., and MEDAWAR, P. B. (1954): Quantitative Studies on Tissue Transplantation Immunity. II. The Origin, Strength and Duration of Actively and Adoptively Acquired Immunity. *Proceedings of the Royal Society*, **143B,** 58.

BURWELL, R. G. (1960*a*): Changes in the First Regional Lymph Node After Homografts and Autografts of Cancellous Bone. *Journal of Anatomy*, **94,** 455.

BURWELL, R. G. (1960*b*): A Study of the First Regional Lymph Nodes Draining Homografts of Red Bone Marrow, Marrow Free Cancellous Bone and Certain Stored Bones. *Journal of Anatomy*, **94,** 575.

BURWELL, R. G., and GOWLAND, G. (1960): Lymph Node Reactivity to Homografts of Cancellous Bone. *Nature*, **188,** 159.

BURWELL, R. G., and GOWLAND, G. (1961): Studies in the Transplantation of Bone. I. Assessment of Antigenicity. Serological Studies. *Journal of Bone and Joint Surgery*, **43–B,** 814.

CARLSON, B., and GYLLENSTEN, L. (1958): Plasma Cells in the Growing Lymphatic System of Young Guinea Pigs. A Quantitative Investigation. *Acta Pathologica et Microbiologica Scandinavica*, **43,** 365.

CARNESALE, P. L., and SPANKUS, J. D. (1959): A Clinical Comparative Study of Autogenous and Homogenous Bone Grafts. *Journal of Bone and Joint Surgery*, **41–A,** 887.

CHALMERS, J. (1959): Transplantation Immunity in Bone Homografting. *Journal of Bone and Joint Surgery*, **41–B,** 160.

CRAIGMYLE, M. B. L. (1958): Regional Lymph Node Changes Induced by Cartilage Homo- and Heterografts in the Rabbit. *Journal of Anatomy* (London), **92,** 74.

DARCY, D. A. (1952): A Study of the Plasma Cell and Lymphocyte Reaction in Rabbit Tissue Homografts. *Philosophical Transactions of the Royal Society of London*, Series B, **236,** 463.

EASTON, T. W. (1952): The Role of Macrophage Movements in the Transport and Elimination of Intravenous Thorium Dioxide in Mice. *American Journal of Anatomy*, **90,** 1.

EGDAHL, R. H., VARCO, R. L., and GOOD, R. A. (1958): Local Reactions and Lymph Node Response to Skin Heterografts Between Rabbits and Rats. *Internal Archives of Allergy*, **13,** 129.

EHRICH, W. (1929): Studies of the Lymphatic Tissue. III. Experimental Studies of the Relation of the Lymphatic Tissue to the Number of Lymphocytes in the Blood in Subcutaneous Infection with Staphylococci. *Journal of Experimental Medicine*, **49,** 347.

EHRICH, W. E., and HARRIS, T. N. (1942): The Formation of Antibodies in the Popliteal Lymph Node in Rabbits. *Journal of Experimental Medicine*, **76,** 335.

ERNSTRÖM, U., and GYLLENSTEN, L. (1959): The Histologic Picture in Thyroxine-induced Lymphatic Hyperplasia. A Quantitative Examination on Guinea Pig. *Acta Pathologica et Microbiologica Scandinavica*, **47,** 243.

FAGRAEUS, A. (1948): Antibody Production in Relation to the Development of Plasma Cells. *Acta Medica Scandinavica*, **130,** Supplementum 204.

FAGRAEUS, A. (1960): Nomenclature of Immunologically Competent Cells. In *Ciba Foundation Symposium on Cellular Aspects of Immunity*. Ed. G. E. W. Wolstenholme and M. O'Connor. London: J. & A. Churchill Ltd.

FISHER, R. A. (1946): Statistical Methods for Research Workers. Tenth Edition. Edinburgh: Oliver and Boyd.

GALLONE, L., RADICI, G., and RIQUIER, G. (1952): Dei reazione dei linfogangli regionali agli omoinneste de pelle. *Archivio ed atti della Società Italiana di Chirurgia*, **2,** 329.

GORDON, S., and HAM, A. (1950): In *Essays in Surgery*, p. 296. Presented to Dr W. E. Gallie. Toronto: University of Toronto Press.

GROBSTEIN, C., and YOUNGNER, J. S. (1949): Combination of Tissues from Different Species in Flask Cultures. *Science*, **110,** 501.

HAM, A. W., and HARRIS, W. R. (1956): Repair and Transplantation of Bone. In *The Biochemistry and Physiology of Bone*. Edited by G. H. Bourne. New York: Academic Press Inc.

HARRIS (1960): In *Ciba Foundation Symposium on Cellular Aspects of Immunity*, p. 120. London: J. & A. Churchill Ltd.

HARRIS, M. (1943): The Compatibility of Rat and Mouse Cells in Mixed Tissue Cultures. *Anatomical Record*, **87,** 107.

HARRIS, T. N., and HARRIS, S. (1949): Histochemical Changes in Lymphocytes During the Production of Antibodies in Lymph Nodes of Rabbits. *Journal of Experimental Medicine*, **90,** 169.

HESLOP, B. F., ZEISS, I. M., and NISBET, N. W. (1960): Studies on Transference of Bone. I. A Comparison of Autologous and Homologous Bone Implants with Reference to Osteocyte Survival, Osteogenesis and Host Reaction. *British Journal of Experimental Pathology*, **41,** 269.

HOLUB, M. (1960): Quoted by Simonsen. In *Ciba Foundation Symposium on Cellular Aspects of Immunity*, p. 118. London: J. & A. Churchill Ltd.

HUTCHISON, J. (1952): The Fate of Experimental Bone Autografts and Homografts. *British Journal of Surgery*, **39,** 552.

KEUNING, F. J., and VAN DER SLIKKE, L. B. (1950): The Role of Immature Plasma Cells, Lymphoblasts and Lymphocytes in the Formation of Antibodies, as Established in Tissue Culture Experiments. *Journal of Laboratory and Clinical Medicine*, **36,** 167.

KOLOUCH, F., GOOD, R. A., and CAMPBELL, B. (1947): The Reticulo-endothelial Origin of the Bone Marrow Plasma Cells in Hypersensitive States. *Journal of Laboratory and Clinical Medicine*, **32,** 749.

LAVIOLETTE, P., and REROLLE, M. (1958): Affrontements *in vitro* de peau de Mammifères adultes. *Comptes Rendus des Séances de la Société de Biologie*, **152,** 610.

LEDUC, E. H., COONS, A. H., and CONNOLLY, J. M. (1953): Immuno-histochemical Localization of Antibodies in the Plasma Cell and its Progenitors. *Anatomical Record*, **117,** 580.

LEDUC, E. H., COONS, A. H., and CONNOLLY, J. M. (1955): Studies on Antibody Production. II. The Primary and Secondary Responses in the Popliteal Lymph Node of the Rabbit. *Journal of Experimental Medicine*, **102,** 61.

MARCHAND, F. (1924): Quoted by Ehrich (1929).

MARSHALL, A. H. E., and WHITE, R. G. (1950): Reactions of the Reticular Tissues to Antigens. *British Journal of Experimental Pathology*, **31,** 157.

MEDAWAR, P. B. (1945): A Second Study of the Behaviour and Fate of Skin Homografts in Rabbits. *Journal of Anatomy* (London), **79,** 157.

MEDAWAR, P. B. (1948): Tests by Tissue Culture Methods on the Nature of Immunity to Transplanted Skin. *Quarterly Journal of Microscopical Science*, **89,** 239.

MEDAWAR, P. B. (1958): The Homograft Reaction. *Proceedings of the Royal Society of London*, **149B,** 145.

MEDAWAR, P. B. (1959*a*): In *Cellular and Humoral Aspects of the Hypersensitive States*. Edited by H. S. Lawrence. London: Cassell.

MEDAWAR, P. B. (1959*b*): In *Biological Problems of Grafting*. Edited by F. Albert and P. B. Medawar. Oxford: Blackwell Scientific Publications.

MEYER, P. C. (1956): The Histological Identification of Osteoid Tissue. *Journal of Pathology and Bacteriology*, **71,** 325.

MITCHISON, N. A. (1954): Passive Transfer of Transplantation Immunity. *Proceedings of the Royal Society*, Series B, **142,** 72.

MITCHISON, N. A. (1955): Studies on the Immunological Response to Foreign Tumor Transplants in the Mouse. I. The Role of Lymph Node Cells in Conferring Immunity by Adoptive Transfer. *Journal of Experimental Medicine*, **102,** 157.

NOSSAL, G. J. V. (1959): Antibody Production by Single Cells. III. The Histology of Antibody Production. *British Journal of Experimental Pathology*, **40,** 301.

OAKLEY, C. L. (1959): The Localization of Antibody Production. In *Modern Trends in Pathology*. Edited by D. H. Collins. London: Butterworth & Co. (Publishers) Ltd.

PEER, L. A. (1954): Cartilage Grafting. *British Journal of Plastic Surgery*, **7,** 250.

RADICI, G., and PIREDDA, A. (1957): La reazione di incompatibilità agli omoinnesti di pelle: aspetti morfologici e significato biologico. Nota I. Modificazioni istologiche dei linfogangli regionali. *Giornale Italiano di Dermatologia*, **1,** 49.

RICH, A. R., LEWIS, M. R., and WINTROBE, M. M. (1939): The Activity of the Lymphocyte in the Body's Reaction to Foreign Protein, as Established by the Identification of the Acute Splenic Tumor Cell. *Bulletin of the Johns Hopkins Hospital*, **65,** 311.

RINGERTZ, N., and ADAMSON, C. A. (1950): The Lymph-Node Response to Various Antigens. *Acta Pathologica et Microbiologica Scandinavica*, Supplementum **86,** 5.

ROBERTS, J. C., Jun., DIXON, F. J., and WEIGLE, W. O. (1957): Antibody-producing Lymph Node Cells and Peritoneal Exudate Cells. *Archives of Pathology*, **64,** 324.

SCOTHORNE, R. J. (1957): Studies on the Response of the Regional Lymph Node to Skin Homografts. *Annals of the New York Academy of Sciences*, **64,** 1028.

SCOTHORNE, R. J., and MCGREGOR, I. A. (1955): Cellular Changes in Lymph Nodes and Spleen Following Skin Homografting in the Rabbit. *Journal of Anatomy* (London), **89,** 283.

SCOTHORNE, R. J., and NAGY, I. (1960): *In vitro* Studies of the Interaction of Lymph Node and Homologous Tissue. *Annals of the New York Academy of Sciences*, **87,** 149.

SHARP, J. A., and BURWELL, R. G. (1960*a*): The Interaction (" Peripolesis ") of Macrophages and Lymphocytes after Skin Homografting or Challenge with Soluble Antigens. *Nature*, **188,** 474.

SHARP, J. A., and BURWELL, R. G. (1960*b*): Tissue Culture Studies of Lymphoid Tissue after Skin Homografting in Rabbits. *Journal of Anatomy*, **94,** 575.

SHEPRO, D., EIDELHOCH, L. P., and PATT, D. I. (1960): Lymph Node Responses to Malignant Homo- and Heterografts in the Hamster. *Anatomical Record*, **136,** 393.

SJÖVALL, A., and SJÖVALL, Helge (1930): Experimentelle Studien über die Sekundärknötchen in den Kniehehlen-lymphknoten des Kaninchens bei Bacillus pyocyaneus-Infektion. *Archiv für Pathologische Anatomie*, **278,** 258.

SUNDBERG, R. D. (1955): Lymphocytes and Plasma Cells. *Annals of the New York Academy of Sciences*, **59,** 671.

TERASAKI, P. I. (1959): Identification of the Type of Blood-Cell Responsible for the Graft-*versus*-Host Reaction in Chicks. *Journal of Embryology and Experimental Morphology*, **7,** 394.

THIÉRY, J. P. (1960): Microcinematographic Contributions to the Study of Plasma Cells. In *Ciba Foundation Symposium on Cellular Aspects of Immunity*. London: J. & A. Churchill Ltd.

UNNO, G., HANAOKA, M., IWAI, H., HASHIMOTO, S., and MORITA, S. (1954): Cytological Studies on Lymphogonia. *Acta Pathologica Japonica*, **4,** 75.

WEAVER, J. M., ALGIRE, G. H., and PREHN, R. T. (1955): The Growth of Cells *in vivo* in Diffusion Chambers. II. The Role of Cells in the Destruction of Homografts in Mice. *Journal of the National Cancer Institute*, **15,** 1737.

WILLIS, R. A. (1950): *The Principles of Pathology*. London: Butterworth & Co. (Publishers) Ltd.

WOODRUFF, M. F. A. (1957): Cellular and Humoral Factors in the Immunity to Skin Homografts: Experiments with a Porous Membrane. *Annals of the New York Academy of Sciences*, **64,** 1014.

IN MEMORIAM

SAMUEL THOMPSON IRWIN, Kt., C.B.E., B.A., M.B., M.Ch., F.R.C.S.(Edin.), D.L., M.P.
1877–1961

Sir Samuel Irwin died in June at his home in Belfast at the age of eighty-three. With his death the United Kingdom, and especially Ulster, has lost an illustrious surgeon of the old school, a politician of integrity, a vigorous supporter of manly sports and an enthusiastic believer in British institutions.

S. T., as he was affectionately known to a host of friends, was born in 1877 at Claudy, Co. Londonderry. He was educated at Foyle College, Londonderry, and Queen's College, Belfast. He graduated B.A. in 1900 in the old Royal University of Ireland and two years later he obtained, with honours, the degrees of M.B., B.Ch., B.A.O. In 1906 he became a Master of Surgery and in 1911 a Fellow of the Royal College of Surgeons of Edinburgh. He was on the staff of the Royal Victoria Hospital, Belfast, for over forty years. Here he practised general surgery, and although he leaned steadily towards orthopaedics he never gave up the general practice of surgery. The idea of specialising in orthopaedics was ever with him but for various reasons he left this to his juniors, who drew strength and support from his interest and experience in this field. His interest in orthopaedics started in 1911 when he was appointed to the Ulster Hospital for Children, a hospital which was to disappear one night in 1941 when Hitler's Luftwaffe attacked Belfast.

During the 1914–18 war he served on the surgical staff of the Ulster Volunteer Force Hospital with the rank of Captain, R.A.M.C. During the 1939–45 war he was chairman of the Northern Ireland Medical War Committee and in recognition of his services was awarded the C.B.E. in 1947.

He was one of the oldest members of the British Orthopaedic Association, one of the British members of La Société Internationale de Chirurgie Orthopédique et de Traumatologie and, nearer home, Vice-Chairman of the Northern Ireland Council for Orthopaedic Development from its inception in 1940.

S. T. was always a hard worker. Retirement from his hospitals brought little respite because he went on serving his community in many ways: as Vice-Chairman of the Northern Ireland Health Services Board, member of the Northern Ireland Regional Hospitals Board, president of the Central Presbyterian Association, and member of the Senate of Queen's University—to mention only a few of his activities.

In 1957, when he was eighty years of age, the Queen conferred upon him a knighthood, a fitting and well earned acknowledgment of his work and position in Northern Ireland. In younger days he was a keen sportsman—a first class cricketer and a rugby international in those days when the thunder of the " Irish hoofs " struck terror into many a stout heart. He played for Ireland on nine occasions from 1900 to 1903, and in 1935 was elected president of the Irish Rugby Union. He played golf regularly until his eightieth year. In 1947–48 he was Captain of the Royal County Down Golf Club where, during the British Orthopaedic Association Meeting in Belfast in 1948, many orthopaedic surgeons were entertained to such a degree that the sailing of the Liverpool boat had to be postponed for several hours. S. T. carried these honours and distinctions with dignity and unaffected humility and to the end he remained an unassuming personality, shy in disposition and often unaware of the love and affection which so many had for him.

But to know him well you had to travel with him and then be entertained in his own home. As one sat beside him in a train, on board ship or in his study he radiated happiness and confidence. His quiet mind, his equanimity, his fund of knowledge of people and affairs, his gentle humour and his insatiable thirst for information made him a companion of rare quality.

This quiet Ulsterman leaves a large family—two daughters and three sons. One of them, Captain J. W. S. Irwin, was taken prisoner of war at Dunkirk, was an Irish rugby international player and is now senior surgeon in his father's beloved hospital, the Royal Victoria in Belfast. The other sons also served in the Forces during the last war. Both are doctors, one in general practice in Andover and the other a gynaecologist in Northern Ireland. There are many grandchildren who were a constant source of joy to S. T.: as he often said, they made up " a mixed hockey team and three subs." This fine family must feel proud that they stem from one who throughout his long and useful life was respected by all and loved by the many to whom he gave freely and generously his friendship, help and guidance. R. J. W. W.

NIKOLAY NIKOLAIEVITCH PRIOROV

1885–1961

With the death of Professor Priorov on April 15, 1961, Russia lost one of her foremost men of medicine—a scientist and pioneer orthopaedic surgeon.

N. N. Priorov was born in 1885 in the town of Shenkursk. He graduated in 1912 from the Medical Faculty of Tomsk University, and completed his training at the Botkin Hospital and other centres in Moscow. Thereafter he devoted his career to orthopaedic and traumatic surgery, and was quickly recognised as an authority on amputations, prosthetics and

rehabilitation. He gained a wide experience from the treatment of servicemen injured in the first world war. He was responsible for the establishment of the Central Research Institution of Traumatology, Orthopaedics and Prosthetics, of which he remained the head until the end of his life. In this capacity he played an important part in the development of orthopaedics in Russia and in the training of junior orthopaedic surgeons. He also held the Chair of Traumatology and Orthopaedics in the First Moscow Medical Institute, as well as other important posts. In the second world war he was surgeon-in-chief of the Evacuation Hospitals of the Public Health Ministry, and later he was appointed Deputy Minister of Public Health.

In the academic sphere Professor Priorov was a prolific worker and contributed more than 200 scientific papers to the surgical journals. He was a founder member and chairman of the All-Union Society of Traumatology and Orthopaedics. He was also a member of the International Society of Surgery and an Honorary Fellow of the British Orthopaedic Association.

B. A. P. AND R. G.

PROCEEDINGS AND REPORTS OF UNIVERSITIES COLLEGES, COUNCILS AND ASSOCIATIONS

GREAT BRITAIN AND IRELAND

BRITISH ORTHOPAEDIC ASSOCIATION

ELECTION OF OFFICERS AND OF MEMBERS OF EXECUTIVE COMMITTEE

At a business meeting on September 29, 1961, the following officers were elected.

Vice-presidents for 1962—Professor Roland Barnes (Glasgow), Mr Norman Roberts (Liverpool).

Honorary Treasurer for 1962-63—Mr J. S. Batchelor (London).

Honorary Secretary for 1962-63—Mr T. J. Fairbank (Cambridge).

Members of Executive Committee for 1962-63—Mr J. G. Bonnin (London), Mr C. H. Cullen (Manchester), Mr G. J. Lillie (Plymouth), Mr Athol Parkes (Glasgow).

ELECTION OF FELLOWS, MEMBERS AND ASSOCIATES

The following were elected.

Honorary Fellows—J. B. Colquhoun (Melbourne), G. E. Godber (Ministry of Health).

Corresponding Fellow—T. G. Garofalides (Athens).

Emeritus Fellows—B. H. Burns (London), R. I. Stirling (Edinburgh).

Fellows—J. G. Bickerton (Durban), N. J. Blockey (Glasgow), R. S. Henderson (Edmonton, Alberta), L. Henry (Cheltenham), H. R. T. Hodgkinson (Sydney), St J. G. O'Connell (Cork), D. P. Rowe (Sydney).

Members—M. Alms (Saskatoon), A. Axer (Tel-Aviv), A. Benjamin (London), G. T. F. Braddock (London), R. A. Bremner (York), J. R. Briggs (London), B. H. Brock (Salisbury), P. I. Busfield (London), D. M. Caird (Middlesbrough), H. Carrier (Chicoutimi), B. Cashman (Bedford), A. K. Cavadias (Athens), E. A. Chisholm (Dumfries), R. S. Cowie (Shrewsbury), P. I. Cromack (Perth, W.A.), M. A. Dimitsas (Athens), P. A. Freeman (Glasgow), P. R. French (London), L. C. L. Gonet (London), C. R. Griffin (R.A.F.), B. J. S. Grogono (Winnipeg), A. J. Harrold (London), R. J. Hart (Cheltenham), J. R. Hutchinson (Burnley), W. G. Kerr (Dar-es-Salaam), P. Kilburn (Liverpool), H. G. Lowe (Stafford), I. McKechnie (Peterborough), I. G. MacKenzie (Aberdeen), D. M. McMillan (Croydon), J. D. Malloch (Scunthorpe), A. H. G. Murley (Cambridge), A. Z. Nsouli (Beirut), J. Piggot (Belfast), A. G. Pollen (Bedford), H. D. W. Powell (High Wycombe), J. B. Pyper (Belfast), A. H. C. Ratliff (Southend), B. F. Regan (Kilkenny), P. A. Ring (Redhill), D. E. Robertson (Saskatoon), R. H. C. Robins (Truro), P. N. Robson (Newcastle upon Tyne), F. W. Shea (Leeds), G. C. Slee (Hereford), F. E. Soutter (Carluke), A. J. Spence (Kirkcaldy), R. L. Stephen (Sydney), I. M. Stewart (Dundee), D. R. Sweetnam (London), R. E. Wherrett (Sydney), I. W. Winchester (London), D. G. Wright (Newcastle upon Tyne).

Associates—J. G. Allman (London), B. G. Andrews (London), C. J. Arapakis (Black Notley), A. Brodetti (Hong Kong), S. Bulos (Beirut), J. N. Cozens-Hardy (London), A. K. Das (Calcutta), A. S. Dawson (Inverness), B. H. Day (London), R. F. N. Duke (Bristol), I. Fletcher (London), V. M. da Costa Garrett (Portuguese Air Force), D. J. Ghadialy (Mauritius), D. S. Grewal (Stoke-on-Trent), A. N. Henry (Bristol), C. H. Hooker (Oswestry), P. J. Kleyn (Cape Town), S. F. Lam (Hong Kong), D. R. Lee (Durban), R. S. M. Ling (Edinburgh), R. K. McRae (Glasgow), P. J. Millroy (London), K. Norcross (Manchester), B. T. O'Connor (Oxford), E. G. M. Palser (London), D. M. Ritchie (Birmingham), J. G. Sallis (London), P. M. Scott (Aberdeen), P. R. Shires (Pyrford), N. N. L. Teckkam (Mauritius), W. A. L. Thompson (Liverpool), R. N. Tinning (Edinburgh), M. Vitali (London).

EUROPEAN TRAVELLING SCHOLARSHIP, 1960

Mr G. C. Slee, who was awarded a British Orthopaedic Association European Travelling Scholarship in 1960, visited accident services in Europe. Mr Slee writes:

SWEDEN

In Stockholm almost all fractures and injuries are treated by the general surgeons. Although the orthopaedic department at the Karolinska Institute is large it does not undertake the early treatment of injuries, which are dealt with by the general surgical team on duty for the day. No special arrangements are made for the treatment of injured patients, who are treated in the general surgical wards.

In one of the larger general hospitals in Stockholm the Department of Surgery has virtually a sub-department of traumatic surgery under the care of Dr Ivor Palmer, who is regarded as the leading traumatic surgeon in Sweden. This is a very well run department, but there are no features of special interest. The junior staff are all young men undergoing general surgical training, most of them without any orthopaedic experience. Dr Palmer himself retires in a few years time. Dr Mac Fellander, the orthopaedic surgeon at St Goran's Hospital, runs the only mixed orthopaedic and traumatic unit in Stockholm and he shares the accident cases with his general surgical colleagues.

In Uppsala, where Professor Carl Hirsch held the chair of Orthopaedic Surgery, the situation was much the same. He had only about forty beds in his unit and dealt with little or no trauma.

Within the city of Göteborg there are three small polyclinics which deal with the smaller injuries and undisplaced fractures of small bones until discharge. More severe injuries and any fractures which require reduction are referred to the Sahlgrenska Sjukhuset Hospital, a beautiful modern and well staffed hospital, where there is a unique department of extremity surgery under Professor Erik Moberg. If an injuried patient can be dealt with as an out-patient he is treated by the general surgeons until discharge. If, however, he has a hand injury or an injury of the extremity necessitating admission, the department of extremity surgery is informed and the patient is admitted. Although this is the agreed arrangement, many patients with injuries of the extremities find their way to the general surgical wards. This is not unrelated to the way in which doctors in the hospital service in Sweden are paid, for they receive payment for each out-patient when first seen and for each operation performed while the patient is in hospital, and for every out-patient attendance after discharge from hospital. The payment is made by the patient, who later obtains a 75 per cent rebate from the Health Service.

During the night, however, the department of extremity surgery is kept quite busy, as often the general surgical registrar on call feels that he would rather remain in his bed than collect the ten or fifteen Swedish crowns which would be his reward for getting up to examine the patient.

This unusual arrangement has come about mainly because this Chair was specially created for Professor Moberg, who is a general surgeon specialising in hand surgery. Apart from fractures of the bones of the hands he is not interested in other " minor fractures " in which he includes Colles' fracture. He thinks that these should be dealt with by the general surgeons because there are not sufficient orthopaedic surgeons to go round. The department of extremity surgery is a very well planned new building and is well organised for its deliberately limited field of activity.

The University Orthopaedic Hospital at Göteborg does not deal with any injuries.

Malmö—All injuries in Malmö go to the General Hospital, which serves a district with a quarter of a million population, and is the only one in Sweden in which all skeletal injuries are dealt with by orthopaedic surgeons. Dr S. von Rosen is in charge of a department of 130 beds. Each department in this hospital is housed in a separate building, and attached to the general surgical building is the emergency admissions department. To this department many of the patients with injuries are brought, though not all of them, others being taken direct to the orthopaedic department, depending entirely upon the ambulance driver or the taxi driver who brings the patient. Often if they know that the patient has a broken bone they will take him straight to the orthopaedic department instead of to the reception. However, the liaison between general surgeons and orthopaedic surgeons appears to be excellent and all fractures and injuries of the locomotive system are referred to the orthopaedic department. Herein lies one of the big drawbacks of the arrangement at Malmö, for it is difficult to transfer patients between the various departments. Even the radiology department is in a separate building, though there are small x-ray machines in both the orthopaedic buildings. Here again is an added complication, for the orthopaedic out-patients are housed in a separate building 200 yards from the in-patients.

In the near future, however, all the surgical specialities are to be grouped together within one surgical block, which should enable reception and treatment of injured patients to be performed more effectively and more rapidly, and the co-operation which already exists between the various surgical departments will be made even more valuable.

Hand injuries are all referred to the department of hand surgery which is part of the department of general surgery. There they are dealt with by Dr Nils Carstam. Malmö has no university, but the hospital is associated with the University of Lund. The overall head of the Department of Orthopaedic Surgery in Lund and Malmö is Professor Gunnar Wiberg, who is stationed at Lund about twelve miles away. There is a department of neurosurgery in Lund, but not in Malmö. Consequently, a minor head injury with other injuries would be sent to the orthopaedic wards, but a moderate head injury with a fracture would be admitted to the general surgical wards and the orthopaedic surgeons would be invited to look after the other injuries; and a severe head injury would be transferred to the neurosurgeons at Lund. Burns are transferred to the plastic surgery department. Peripheral nerve injuries in the upper limbs are usually dealt with by the department of hand surgery, but in the lower limbs peripheral nerve injuries are referred straight to the orthopaedic surgeon.

In Professor Wiberg's department in Lund the orthopaedic department deals with a little over 50 per cent of bone and joint injuries. The arrangement there is that the orthopaedic department is on call for treatment of these injuries on alternate days, but all fractures into joints are referred to them.

Lund is an old university town somewhat similar to Oxford or Cambridge. It is not large and is at the moment suffering from a shortage of nurses, so severe that it has been the subject of headline news in the Swedish newspapers. Already two of the orthopaedic wards are closed down. There is a possibility of further closing of wards which would mean that a great number of injuries would have to be transferred to Malmö.

Generally speaking, the orthopaedic surgeons in Sweden would very much like to be in charge of the treatment of injuries to the musculo-skeletal system. The chief difficulty lies in the training of the young orthopaedic surgeons in traumatic surgery, for the only places in Sweden where trauma is dealt with by the orthopaedic surgeons are Malmö and, to a lesser extent, Lund. Apart from these two centres, young orthopaedic surgeons have to obtain their accident surgery experience in general surgical units, and with the exceptions of Palmer's unit in Stockholm and Moberg's in Göteborg, the standard of fracture treatment by the general surgeons is not considered to be very high. In addition there is an acute shortage of doctors in Sweden. Most doctors in the hospital service work in the large cities in the south, and the vast expanse of northern Sweden where there are many small towns but no large cities finds great difficulty in staffing its hospitals. Thus in a small Swedish town the surgeon is frequently the only one for many miles and has to undertake all emergencies.

There is no definite organisation for the treatment of traumatic paraplegia and no centres for paraplegics in Sweden yet. Some such patients are treated in neurosurgical wards, a few in the orthopaedic wards and the rest in general surgical wards.

DENMARK

In Denmark the situation is very much the same as in Sweden; almost all injuries are dealt with by general surgeons at the same time as, and beside, the general surgical patient. There are no hospitals with any specialised traumatic units. The Orthopaedic Hospital in Copenhagen received emergencies on Thursday only and that in Aarhus does not deal with any fresh injuries at all. At the moment there are only six or seven hospitals outside these two cities where orthopaedic surgeons are available, and here again, as in Sweden, the adequate training of orthopaedic surgeons is a problem.

GERMANY

I spent only a few days in Hamburg and Munich and visited only the orthopaedic hospitals in these places. Fractures are treated by the general surgeon in Germany.

AUSTRIA

Austria is the only country in Europe with a well organised series of accident hospitals, and this is the result of private enterprise on the part of a large insurance company, the "Allgemeine Unfallverischerungs Anstalt" (abbreviated to A.U.V.A.). The evolution of this state of affairs is interesting. Lorenz Böhler was appointed in Vienna to deal with injuries in the university surgical clinics, with the agreement of the general surgeons in principle, though not in practice, because they refused to allow him the use of any beds. A.U.V.A. were delighted that at last someone was going to take a special interest in accident surgery, because they felt that too many workers were being permanently crippled as a result of industrial accidents, and so they offered to build a hospital for Böhler. Thus the original Unfallkrankenhaus of Vienna, in Webergasse, was opened in 1926 in a building which had originally been designed as a bank and offices. It eventually became the prototype

for the other accident hospitals later established by A.U.V.A. in Graz, Linz, Salzburg, Klagenfurt and a second one in Vienna. Since the new hospital was opened in Vienna in 1955 the beds in Böhler's original Unfallkrankenhaus have been reduced from 150 to seventy-five, the insurance company expanding its administrative side into the other half. Working in conjunction with these accident hospitals are a number of country rehabilitation hospitals also established by A.U.V.A. All these hospitals are completely independent of the Austrian State Medical Services.

I visited the accident hospital at Linz which is directed by Dr Jorg Böhler—son of Professor Lorenz Böhler—and the old and new hospitals in Vienna. The Linz and the New Vienna hospitals are very similar in design and function and represent the final design arrived at from the experience of a quarter of a century. They are exceptionally well staffed, and in comparison with other Austrian hospitals, their medical staffs are well paid. The new Unfallkrankenhaus in Vienna, under the directorship of Dr Otto Russe, has 212 beds and twenty-six doctors, and at Linz there are 200 beds and twenty-five doctors. With the exception of two anaesthetists the doctors are all accident surgeons, either trained or in training.

To qualify as a specialist in traumatic surgery, an Austrian doctor has to train for at least six years as follows: two and a half years of general surgery, six months of " cold " orthopaedic surgery, and not less than three years of comprehensive traumatic surgery, of which two years have to be spent as an assistant, that is, in a position of responsibility. After one year of traumatic surgery it is not easy to get an assistant's appointment, and consequently it usually takes seven or eight years to fulfil the requirements. During these years of training they have to perform a certain number of various types of surgical procedure and plaster techniques. They all have to be able to give anaesthetics, for they frequently have four or five theatres in operation at the same time, and there is only one specialist and one trainee anaesthetist on the staff. They are organised into three teams who are on duty for twenty-four hours at a time, and sleep within the hospital whilst on call.

The Director is provided with an extremely spacious and comfortable flat in the hospital and is thus almost always available for consultation. Professor Böhler at seventy-five years of age still lives in his hospital, his only concession to the passing years being to spend the week-ends at one of the country rehabilitation hospitals where he " idles " away his leisure hours writing yet another edition of his classical work on fractures.

In hospital design and in the detailed organisation of the day to day work of the hospital there is much which we could usefully incorporate into our own accident services in the future. The ground floor is occupied by the administrative and insurance sections, together with inquiries, telephone exchange, medical records, hospital shop and a very large comfortably furnished waiting room with a buffet bar in one corner. The only medical features of this floor are the out-patient follow-up department and the rehabilitation or physiotherapy department. Reception, diagnostic and treatment areas are usually all on the first floor. All the rooms open on to one long wide corridor, the theatre block at one end being shut off to all but staff by large swing doors. Septic cases requiring operation are dealt with in a special theatre which is situated either on another floor or at the opposite end of the first floor from the " clean " theatres.

Every room in the hospital is numbered in sequence and the number displayed boldly upon the door. Patients are directed to " room number 106 " or " 107 " instead of to the " x-ray department," " plaster room," and so on.

Ambulant patients attending for the first time arrive at the reception room on the first floor where a secretary types the name and address and other particulars straight on to the front sheet of the notes. She also takes the history of the accident unless this is too complicated—when it is taken by one of the examining doctors. The patient now goes into a waiting room until called—over a loudspeaker—into the next room beyond which is the examination room. Meanwhile, his completed front record sheet passes by conveyor belt beneath the floor of the waiting room and into the examination room, and is already in place on the typewriter by the time the patient is called in for examination. He is then examined by one of the two doctors on duty there, who dictates his clinical findings and diagnosis (if made at that stage) to the secretary who types it out immediately. A carbon copy of the particulars of the patient serves as a universal request and order form and is taken with the patient to the radiology department, plaster room, suture theatre, septic theatre, or Unna's paste room as necessary. All these rooms are adjacent. Only very small dressings are done in the examination room itself. The patient returns with his radiographs which are then seen, the report dictated on to the case sheet, the diagnosis completed, the line of treatment indicated and the patient sent to the appropriate treatment room, and then returns home. Alternatively, he may be admitted and dealt with as an in-patient.

The ambulance bringing the severely injured stretcher patient hoots a signal as it approaches the drive-in at the front of the hospital and the electrically activated glass doors slide open allowing it to drive straight inside. The patient and stretcher are lifted on to a trolley which is taken by a

lift to the examination room on the first floor. After a rapid examination and assessment the patient is wheeled down to the theatre area for resuscitation, more detailed assessment—including radiography —and treatment.

The plaster rooms, radiology department, suture theatres and four main operation theatres with resuscitation area are all on the same side of the corridor and all intercommunicate so that patients on trolleys can pass to and fro. Ambulant patients move between the various rooms along the main corridor.

On the patient's next attendance at the hospital he goes to the reception room on the ground floor and his case folder is obtained. If he is in plaster he then goes to the first floor where he is examined and treated in a room for that purpose, which is also used for the daily review of radiographs. Here again all notes are typed from dictation. Shorthand is only used on ward rounds and dictaphones are used at night. If the patient is not in plaster he remains on the ground floor and is examined in one of the follow-up rooms there.

The medical records system is first class and is the British Banda system. The original front record sheet with all the details of the first attendance is printed in whole or in part on to all the necessary hospital medical, administrative and insurance documents, as well as on to the front of the radiograph envelope. Thus the particulars of each patient never have to be written again. A standardised letter to the patient's private doctor carries a full copy of the front sheet.

All the doctors are full time employees and only the directors of the Accident Hospitals are allowed private practice, which is largely orthopaedic in nature. Each day's work starts at 8 o'clock in the morning with a review of all the previous day's radiographs, to which come most of the staff; then teams of surgeons operate, do ward rounds or see out-patients. Every type of injury is dealt with, including those of the head, chest, abdomen, facio-maxillary area, renal tract and so on. I discussed with Dr Jorg Böhler and Dr Otto Russe the vexed question of the competence of any one man to deal successfully with injuries affecting all these various systems. Both held the view that while they personally felt able to cope with the great variety of injuries which presented, it was too great a responsibility for one man, and it was better shared with highly trained specialists, especially in neurosurgery and maxillo-facial, thoracic, and ophthalmic surgery. Dr Böhler believed strongly that the accident service should be part of a general hospital and should benefit from all the facilities thereof, and from the ready co-operation of other specialists. He did not consider that there is any place for a purely accident hospital—even in a large city. On the other hand, Russe thought the accident hospital to be a satisfactory arrangement—as long as the opinions of visiting specialists can be readily obtained.

None of the accident hospitals has radiologists; indeed, Russe considers it essential that the accident surgeon maintains full control over his own radiology department.

Paraplegics are nursed in the accident hospitals and later go to the rehabilitation hospitals—but the traumatic surgeons believe that they should be treated altogether in special centres. One of them told me that if he became paraplegic he would want to be treated in Sheffield or Stoke Mandeville.

Septic cases are segregated in special wards, this being made easier because, as in most hospitals built during this century in Europe, the wards consist of one to four—or occasionally six—beds. This adds considerably to the time taken by ward rounds.

The annual turnover of patients at the new Unfallkrankenhaus in Vienna is 35,000 new patients a year, of which 5,000 are in-patients.

FRANCE

Paris—Much of the traumatic surgery in Paris is carried out by orthopaedic surgeons. Most of the units there, and certainly all those I visited, deal with both orthopaedics and injuries side by side.

The most modern department is that of Professor R. Merle d'Aubigné at the Hôpital Cochin. It was only opened towards the end of 1959 and is very well appointed. It has a basement and six floors as follows: *Basement*—Electrotherapy and physiotherapy, photography, radiology and laboratory. *Ground floor*—Out-patients with reception, records, emergencies department with theatre and plaster room. Also facilities for teaching, including library and documentation room, waiting hall, lecture and conference room. *First floor*—Operation theatre suite with viewing galleries above and wards. *Second, third and fourth floors*—Wards, includes an air-conditioned burns unit on the fourth floor. *Fifth floor*—Doctors' accommodation.

Walking and stretcher patients enter the same central doorway, cross a large hallway where inquiries, lifts, staircases and telephone exchange are situated, and go straight into the emergency department. Ambulances drive up a ramp and unload under complete cover. Orthopaedic and fracture out-patients enter through a separate doorway. Lifts, conveniently situated to the emergency department, take patients up to the wards or down to the radiology department. There is no special resuscitation room.

The theatre suite was the most impressive I saw in Europe, or, for that matter, in those hospitals I visited in Canada and the United States in 1956. There are four hexagonal theatres laid out in a square pattern, with scrubbing up facilities located centrally and sterilising facilities between each pair. There is a glass dome over each theatre which projects into the floor above and around this is a rail and benches for spectators, who look down—quite literally—into the field of operation. I have never before had such an excellent view of operations in which I was not personally taking part. The lighting is situated outside the theatre above the glass dome, and consists of a large battery of multiple lights shining at various angles with a central, independently mobile part. Thus two separate areas can be illuminated simultaneously. They are electrically operated from the theatre below. The surgeon wears a throat or chest microphone and can give a commentary to the spectators' gallery through a loudspeaker.

In the theatres themselves all anaesthetic gases are piped in through floor standards, all doors are of glass and are moved electrically by foot switches; and instrument packs, towels and dressings are passed in through a service hatch in one wall from a sterilising and preparation room common to two theatres. Each theatre is irradiated by six ultra-violet light strips on the walls. One theatre has a battery of floodlights occupying the whole of one wall; and there is a platform for the cameras and cameraman, fixed on the mobile lights outside the dome overhead.

This theatre suite should be visited by anyone planning theatre accommodation for a new teaching hospital. The layout is good for a mixed orthopaedic and accident unit, but suffers from being separated from the general surgical and neurosurgical blocks which are some distance away. Unfortunately I was not able to get figures of the numbers of accidents treated here, but thought that they seemed but a small part of the work of the whole unit.

The Hôpital St Louis is one of the oldest hospitals in Paris. It is overcrowded and not very well appointed. Its arrangements for the reception and treatment of accidents are unexceptional. Dr Jean Cauchoix is in charge of the orthopaedic department.

Lyon—The interest here was in the entirely different arrangements for accident reception.

The Hôpital Edouard Herriot has almost 3,000 beds and is the largest in the city. There are about six other hospitals, all of which deal with injuries during the day, but at night most are taken to Hôpital Edouard Herriot and an occasional one to the Hotel Dieu. During the day ambulances take any obvious or suspected bony injuries directly to the Centre de Traumatologie which is directed by an orthopaedic surgeon.

Multiple injuries are taken direct to the emergency block, where there is a surgeon on duty who is responsible for triage. Emergency treatment is given and the patient transferred as soon as possible to the appropriate traumatic, thoracic, neurosurgical service.

From 4 o'clock in the afternoon until 9 o'clock on the following morning any surgical or medical emergency arriving at the hospital is admitted to the emergency block which is near the main gates; but obstetrical or gynaecological emergencies are taken to a separate department which stands opposite.

The emergency block is under the administrative charge of an orthopaedic surgeon, Dr A. Trillat. There are one hundred beds available, of which he is allowed twenty for orthopaedic patients. Of the remaining eighty beds there have to be forty to sixty empty by 4 o'clock each afternoon to be ready to deal with admissions during the night, when the block is staffed by two surgeons—from a rota of twenty-six—who do a week's tour of duty at a time; they include assistants from all the surgical departments in the hospital, such as the general surgical, orthopaedic, neurosurgical, ear, nose and throat, thoracic, facio-maxillary and so on. All of them are expected to be able to deal with most medical and surgical emergencies, but they can call upon the appropriate consultant for assistance in difficulty. They are also responsible for moving patients to the appropriate department during the first few days after admission. Thus Dr Trillat, the surgeon-in-charge of the emergency block, has virtually nothing to do with the eighty emergency beds unless he has been called in as an orthopaedic opinion by the surgeon on night duty.

Dr Trillat agreed with me that this system of emergency admissions was not very satisfactory, especially from injured patients' point of view. He and his colleagues had tried to have the system altered so that all fractures were taken straight to the traumatic centre throughout the twenty-four hours, but the administration consider the present system the most economical.

This huge hospital is yet another of those in which each department is situated in its own completely separate building. Another orthopaedic service, that of Professor M. Guilleminet, which includes children but no accidents, is situated in yet another building. All the buildings are connected, however, by an extensive network of subterranean passages along which electrically propelled trucks and trolleys move rapidly and quietly on rubber-tyred wheels carrying food, stores, supplies and patients from one building to another.

The traumatic centre has been recently considerably altered internally and has a new twin theatre suite the design of which was based on that in the Hôpital Cochin in Paris. The layout is reasonably good, but unfortunately is spoilt by shortage of space throughout.

In addition to these accident centres I visited many other orthopaedic clinics in the countries of Western Europe during my fourteen weeks' tour. I saw much to think about and much to tell about. I was treated with great kindness everywhere, and with considerable patience. I feel more of a European now and value highly my memories of those surgeons whom I met. I shall always be grateful to the British Orthopaedic Association for giving me this splendid opportunity.

SCIENTIFIC SOCIETIES AND REGIONAL ORTHOPAEDIC CLUBS

THE ORTHOPAEDIC TRAVELLING CLUB

The first meeting of the Orthopaedic Travelling Club was held in Copenhagen, Malmö and Lund from May 29 to June 3, 1961. In Copenhagen the meeting was held at the Orthopaedic Hospital where the proceedings were opened by Professor Arne Bertelsen.

Muscle regeneration: incidence of scoliosis and foot deformities after poliomyelitis—*Dr Niels Bukh* (Viborg) described the findings in 240 patients suffering from poliomyelitis. Regular muscle testing had been carried out from six weeks to five years after onset. Fifty per cent of the ultimate recovery had taken place within six months, 80 per cent in one year and 95 per cent in two years. The degree of recovery was in direct proportion to the quality of the surrounding muscles. Back muscles recovered more rapidly than abdominal muscles. He had followed up 508 patients in regard to foot deformities and scoliosis for five years. One-half of the foot deformities originally noted had corrected within five years. In scoliosis imbalance of trunk and psoas muscles promoted deformity but psoas imbalance alone did not. It was unusual for deformity to develop in patients over fifteen years of age. *Professor J. I. P. James* (Edinburgh) said that scoliosis did not develop after skeletal maturity. High thoracic scoliosis was due to intercostal paralysis. *Mr Norman Roberts* (Liverpool) said that it was important in such a review to break down the recovery rates into children or adults. *Dr O. M. Hansen* (Copenhagen) said that, in muscle recovery, nerve end plates were enlarged and supplied more fibres. Dr Bukh replied that he had not found that scoliosis developed after seventeen years of age. He found that recovery in children was more rapid in the first six months but after that it was more rapid in adults. The difference was small. He had not found that training of muscles made any appreciable difference to the recovery, but prevention of deformity was important.

Experience with the Grice operation for the paralytic foot after poliomyelitis—*Dr L. Zachariae* and *D. H. Moller* (Copenhagen) described their experiences with the Grice bone block operation for valgus feet. Sixty-nine patients had been treated. Forty-six were good and twenty-three were poor; the foot was in too much valgus in twenty and in too much varus in three; but none had pain. Instability of the ankle joint was a difficult problem which might ultimately necessitate a pan-talar arthrodesis and a tight calcaneal tendon might prejudice the result. Muscle transplants could be combined with the operation with advantage.

Steindler's tendon transfer for paralytic elbow—*Dr K. Nyholm* (Copenhagen) reported the results of Steindler's tendon transfer for paralytic elbow in twenty-three patients. The operation should not be done in persons under fourteen years of age. The displacement should always be as high as possible, and stabilisation of the shoulder greatly improved the results. In only six was flexion full, and in all of these the shoulder had been stabilised.

Tendon transfers in forearm and hand for paralytic disorders—*Dr K. Bang Rasmussen* (Copenhagen) described the findings in 128 patients with paralysis of the hand in whom tendon transfers had been done. He had had no experience of the Brand operation but had found other operations to replace the lumbrical and interosseous muscles to be unsatisfactory. For loss of opposition of the thumb he did not like the Bunnell or Thompson operation but preferred that of Riordan. The use of a sling from the pisiform was essential to prevent the tendon from displacing proximally. Arthrodesis of the first and second metacarpals was a good operation, and if paralysis was more extensive arthrodesis of the metacarpo-phalangeal or interphalangeal joints might be required.

Life-threatening respiratory paralysis—*Professor H. C. A. Lassen* (Copenhagen) described his experience during the poliomyelitis epidemic in 1953. Eight to ten patients with respiratory paralysis were being admitted each day and at one time seventy patients were on artificial respiration. Experience with tracheotomy had enabled them to reduce the mortality rate from 87 per cent to 11 per cent. A total of 350 patients had been treated, of whom two-fifths had died. In the six years since then 195 patients with other forms of respiratory paralysis had been treated. Many of these were suffering from tetanus. He showed a film of the hospital care during the acute phase and of the rehabilitation for home life

afterwards, and also the facilities provided for these patients and their families so that they might lead lives as nearly normal as possible.

Chronic respiratory paralysis after poliomyelitis—*Dr J. Mortens* and *Dr B. Sury* (Copenhagen) reported the outcome of 250 patients treated during the epidemic of poliomyelitis for life-threatening respiratory paralysis. One hundred and ninety-two were still alive. One hundred and seventy-one had very largely recovered from the respiratory paralysis but some had other residual disabilities. Twenty-one still needed positive pressure respiration, fourteen continuously and seven intermittently. All had tetraplegia, in some combined with severe deformities.

Some aspects of poliomyelitis—*Dr O. Remvig* and *Dr B. Eskesen* (Hornbæk) had reviewed thirty-two adults with paraplegia after the poliomyelitis epidemic in regard to physical and social rehabilitation. Paralysis was permanent in all, but in none was the upper limb involved. The average length of in-patient treatment was sixteen months. Twenty-nine reached the stage of self-care in two years, fourteen were functional walkers, fifteen used wheel-chairs as well as walking and three could not walk. Twenty-one had returned to full-time work, five were doing part-time work and six were doing no work. The previous standard of education played a big part in the prognosis of rehabilitation. *Mr J. M. P. Clark* (Leeds) discussed the principles of tendon transfer. Deformity must first of all be corrected. Muscle transfer for loss of power was practical only in the upper limb, and the more distal it was the better. The time for operation was when no further recovery could be expected. Among successful transfers in the upper limb were transfer of pectoralis minor for winged scapula, of pectoralis major to restore flexion of the elbow (this was most satisfactory when the forearm muscles were already producing some flexion), flexor-extensor transfer for drop wrist, and transfer to restore pronation of the thumb. In the lower limb the object of muscle transfer was to achieve balance. Sometimes it must be combined with bony correction, such as osteotomy of the calcaneum or Grice's bone block. Among satisfactory transfers in the lower limb were multiple transplants into the heel for calcaneo-cavus and psoas transplant in paralytic dislocation of the hip. *Mr Robert Roaf* (Oswestry) said that deformity in poliomyelitis might be primary or secondary. For instance, the rib deformity in scoliosis was usually secondary to the deformity of the spine. Unequal muscle pull might cause abnormal joint posture but by itself did not cause bone deformity. In a survey of upper limbs affected by poliomyelitis, spastic paralysis and Erb's palsy, bony deformity was found only when some other factor operated. In children who used crutches cubitus varus due to inhibition of the growth of the trochlea was common. After prolonged splinting in abduction 90 degrees of rotation of the head of the humerus might develop, and after a period of hyper-pronation or supination of the forearm dislocation of the head of the radius might occur, but bony deformity due to muscle pull alone was never seen in his series. If it was agreed that some other factors, such as an abnormal gait or weight bearing, were necessary for bony deformity to occur this would explain the onset of scoliosis and calcaneus deformities.

Accident services—A discussion was opened by *Mr H. Osmond-Clarke* (London). He said that he was chairman of a committee comprised of representatives of orthopaedic, general, plastic and neurological surgeons and general practitioners. A considerable measure of agreement had been obtained. The present defects were the absence of any regional organisation, too many departments, poor facilities and not enough staff. They had advised a comprehensive accident service on a national scale, with provision for all types of injury. Such service was not possible in every hospital. An accident area would comprise a population of one to one and a half million. This would be based on a central hospital capable of dealing with special injuries requiring forms of treatment not available in the average general hospital. This would be attached to a teaching hospital if possible and would encourage research. More peripherally would be four or five accident services based on a general hospital. These units would be staffed and equipped to deal with all but the exceptional case. Smaller units equipped to deal only with minor injuries would be situated in the smaller towns and would be staffed by general practitioners. *Professor A. Bertelsen* (Copenhagen) said that the problem of accidents was not so urgent in Denmark. The acceptance of orthopaedic responsibility for the majority of accidents was not so well advanced as in Britain. Orthopaedic facilities for the treatment of fresh fractures were limited, but the sequelae of fractures were treated in orthopaedic hospitals. *Dr G. Bauer* (Malmö) said that in Sweden the peripheral hospitals were mainly general, and orthopaedic surgery took only a small part. In most the treatment of fresh fractures was the responsibility of general surgeons. It was hoped to increase the orthopaedic responsibility. The central hospital was equipped and staffed to deal with all orthopaedic and accident contingencies and was usually the centre of a population of one and a half million. The problem of who should be in charge of patients suffering from multiple injuries had been discussed for years, but in fact such cases were rare. *Mr Norman Roberts* (Liverpool) said that twenty-five beds per 100,000 population was likely to be insufficient.

Dr Bauer said in Sweden the number was sixty-eight per 100,000 but twenty of these were for orthopaedic surgery.

Perthes' disease—*Dr O. Remvig* and *Dr H. Mose* (Hornbæk) recounted briefly the history of this condition, which was first described in 1909. The results recorded in the literature showed such wide discrepancy that it was necessary to carry out some controlled review. They had reviewed 257 hips affected by Perthes' disease in 219 patients. Seventy-eight had been treated by strict rest in bed, seventy by " mobile " rest in bed and seventy-one with patten-ended calipers as out-patients. Good results were obtained in 130 (55 per cent), fair in thirty-nine (18 per cent) and poor in seventy-one (27 per cent). The results were estimated on the ultimate shape of the head. There was little to choose between those treated by strict or mobile bed rest, but patients in both these groups were significantly better than those treated as out-patients. The younger the child at the time of onset the better was the ultimate result. *Mr H. Jackson Burrows* (London) said that in the early stages slight flattening of the femoral head was to be seen. Focal bone necrosis occurred and there was a corresponding thickening of the articular cartilage. Subluxation could be demonstrated by measuring the distance between the neck and the bony pelvic brim. *Mr Norman Roberts* (Liverpool) said that the value of bed rest in treatment had been clearly demonstrated. Perhaps all older children should be treated thus, but the younger ones could be treated as out-patients in calipers. *Mr E. W. Somerville* (Oxford) said that it was possible to detect different degrees of Perthes' disease according to the extent to which the blood supply was affected. Firstly, there was the type in which only the lateral part of the capital nucleus was affected, the medial part remaining normal so that any deformity of the affected part led to incongruity in the shape of the head. Secondly, the whole nucleus was affected, was largely absorbed and was replaced by multiple nuclei which fused and formed a slightly flattened but congruous head. Thirdly, there was the type in which the changes were transient and recovery relatively rapid. In the first type only the lateral vessels were damaged by local injury, but in the second type all the vessels were obliterated, possibly by a tense effusion, and in the third type the cause of tension was only temporary. When subluxation occurred it could be corrected by osteotomy. Sometimes the changes in the head did not appear until many months after the initial symptoms. It was difficult to know just how long to observe " observation " hips. *Mr E. A. Nicoll* (Mansfield) said that the possibility of Perthes' disease starting with an acute synovitis interested him. He thought this cause was not uncommon. He asked if there was any evidence of premature fusion of the epiphyses. *Mr F. W. Holdsworth* (Sheffield) asked if it was possible to differentiate in an epiphysis between cartilage, which would ultimately ossify and cartilage which persisted as articular cartilage. *Dr P. Lutken* (Aalborg) described the growth of the ossific nucleus from the surrounding cartilage and the epiphysial plate. Dr Mose replied that synovitis was found in 10 per cent of cases. He had found no evidence of premature closure of the epiphyses. Dr Remvig said that in his opinion all the younger patients and those with both hips affected should be treated in bed.

Surgical anatomy and treatment of patients with arthrogryposis—*Dr O. M. Hansen* (Copenhagen) described his experiences with seventy children suffering from arthrogryposis. Treatment should be started early. Correction by plaster should be started first but early surgery might be necessary. Deformities involved all soft tissues from the skin to the joints. In the muscles much fibrosis was present, which almost suggested that some previous disease had healed. Physiotherapy alone did not improve joint function. He had not found mental intelligence to be reduced, but there was a familial tendency. When one hip was dislocated operation was worth while, but not when both hips were affected.

Congenital dislocation of the hip—*Dr Sophus von Rosen* (Malmö) said that in Malmö, with 240,000 inhabitants, 99 per cent of children were born in the same hospital. All newborn children were examined by an orthopaedic surgeon within three days of birth. Over a period of seven years thirty-nine congenital dislocations of the hip had been diagnosed by means of the Ortolani click test and confirmed by radiographs, for which the hip was abducted 45 degrees and medially rotated. If a dislocation was present the line of the femur pointed towards the opposite anterior superior iliac spine. A simple malleable splint was described which held the legs in the Lorenz position and could be kept on without bandaging. The splint was not removed for four to six weeks and the splintage was continued for three months. Only three patients had not responded to treatment and in all of these the period of splintage had been too short. He doubted if congenital dislocation of the hip was due to any dysplasia; it was due rather to laxity of the joint ligaments which might be secondary to the maternal hormones. Evidence of hormonal disturbance was being obtained but it was still too early to be certain. *Mr E. W. Somerville* (Oxford) said that in the Oxford region the problem of early diagnosis was complicated by the fact that the children were born in many different places and the condition was relatively rare. He asked if Dr von Rosen thought that a form of nursing could be devised which, by preventing

extension of the hips for a period after birth, would prevent stretching of the capsule, and therefore instability. Dr von Rosen replied that he had not considered this and thought we must rely on early diagnosis by training all who saw newborn infants. *Mr T. J. Fairbank* (Cambridge) asked if the click test had ever been positive with a normal radiograph. Dr von Rosen replied that he had never found this.

The place of intramedullary nailing in the treatment of fractures of the femur or tibia—*Mr F. W. Holdsworth* (Sheffield) said that most simple fractures united with simple immobilisation. Those in which internal fixation would be most valuable were those in which it was technically difficult to obtain. He described 366 fractures of the tibia treated with simple immobilisation. Bone grafting was done in 2·5 per cent. Internal fixation was indicated only for malignancy or when extensive plastic surgery was required. In fractures of the femur a nail was helpful if other conditions made immobilisation difficult, if the fracture was in the uppermost third of the shaft—where reduction was difficult, or for non-union. *Mr J. Charnley* (Manchester) said that open reduction delayed callus formation but the power of a fracture to unite was determined at the time of the fracture by the degree of soft-tissue damage and the natural stability of the fracture. In the femur a large nail should be used to obtain immediate fixation and early knee movement. It was difficult to put a large nail into the tibia, and he used a thin one to maintain position only. He advised delaying operation for two to three weeks because this improved the chances of early union. It was better not to open the fracture site. He described a flexible reamer for use in the tibia. *Dr Per Edwards* (Malmö) said that in femoral fractures the nail must provide good fixation, there must be no comminution and the fracture must be in the upper two-thirds of the shaft. In a series of 1,003 fresh femoral fractures so treated there had been forty-six technical complications. In tibial fractures he preferred to use two Rush nails, one introduced from above and the other from below. Devitalised overlying skin was an indication for internal fixation.

Osteoporosis—*Dr G. Bauer* (Malmö) said that osteoporosis was unrelated to hyperparathyroidism. In 150 fractures of the femoral neck seen in one year, only two were suspected of any bone disease. In women, but not in men, the incidence of femoral neck fractures doubled in number for every five years after the age of forty. The same held good for fractures of the lower end of the radius, but began fifteen years later. Hormonal treatment was now in doubt and there was no evidence that it produced any benefit. Bone tracers were being used to obtain evidence about production and absorption of bone in osteoporosis. *Mr H. Jackson Burrows* (London) pointed out that anabolic steroids improved calcium balance as did an increased absorption of calcium.

The surgery of primary osteoarthritis of the hip—*Mr N. Capener* (Exeter) said that, although it was difficult to prove, it was probable that most osteoarthritis of the hip after the age of fifty was primary. There were many paradoxes. The question of hyperaemia versus avascularity was undecided. Why did arthritis not develop in paralysed joints? An operation, to be successful, should be done before the muscles wasted, but who would want a major operation such as arthroplasty, arthrodesis, pseudarthrosis or osteotomy for only slight symptoms. The biology and not the biomechanics should be studied. Cartilage regeneration after osteotomy was difficult to assess but he was able to show three examples. *Mr K. I. Nissen* (London) said that in the early stage of primary osteoarthritis passive flexion of the hip was to 90 degrees or more and the femoral head was still spherical; the cancellous bone was still hyperaemic and capable of repair and long survival. High femoral osteotomy banished pain, preserved movement and arrested the arthritis. Internal splintage was essential. If this were clearly understood the number of hips requiring radical surgery would soon be reduced. *Professor G. Wiberg* (Lund) said that smaller operations should not be forgotten. Obturator neurectomy gave some relief; drilling was helpful in 50 per cent for two to four years and the Pauwels osteotomy was often satisfactory for relieving pain. *Dr K. Jansen* (Copenhagen) said that osteoarthritis was not an entity. Drilling gave relief in 50 per cent but no change took place in the joint. Judet's prosthesis failed because the acrylic became pulverised; Vitallium was better. The prosthesis came loose because of vascular changes in the bone. Arthrodesis was good when the osteoarthritis was unilateral in younger patients. When osteotomy was done the lateral rotation should not be corrected. *Dr Sophus von Rosen* (Malmö) supported the use of Pauwels osteotomy and demonstrated a patient in whose hip great improvement had been obtained. Mr Nissen said we should discard the mechanical concept of osteotomy; its important effects were biological. Professor Wiberg thought both factors played a part. *Mr J. Charnley* (Manchester) said there was no proof of the presence of hyperaemia. In the femoral heads he saw at operation avascularity predominated. If hyperaemia were present arthrodesis in the elderly would be easier. He pointed out that the exposed part of the femoral head was not covered by cartilage. *Dr G. Bauer* (Malmö) said that study with isotopes suggested great osteoblastic activity in the femoral head. *Mr Norman Roberts* (Liverpool)

supported Mr Nissen's views and was opposed to any delay in operation, but he doubted the hyperaemia of bone. *Professor R. Barnes* (Glasgow) asked why it was that 20 per cent of osteotomies failed. Mr Nissen replied that the main reason was procrastination; too many cases were allowed to reach the stage of terminal collapse of the joint.

Slipped upper femoral epiphysis—*Professor G. Wiberg* (Lund) emphasised the importance of early diagnosis. An advanced degree of slip should be corrected. Thirty-two per cent of those with a slight slip, and 92 per cent of those with a severe slip, developed osteoarthritis. When the deformity was fixed, correction could be by subtrochanteric or cervical osteotomy. The latter gave better results but had the risk of producing avascularity of the femoral head. Out of twenty-four cervical osteotomies for a slip exceeding 50 per cent, twenty were good and four had avascular changes. This was the best operation provided the incidence of avascular changes could be kept down to 15 per cent, but, if not, subtrochanteric osteotomy was to be preferred. *Professor A. Bertelsen* (Copenhagen) described his experience with 158 patients with 192 slipped upper femoral epiphyses. When only a slight slip was present reduction was unnecessary; for fixation Moore's pins were preferred to a solid nail. Acute slips should be corrected under general anaesthetic and the epiphysis pinned. Late severe slips required cervical osteotomy which he considered to be the best operation in spite of the risk of avascularity. *Mr D. Wainwright* (Newcastle-under-Lyme) emphasised the importance of early diagnosis. He used Moore's pins for internal fixation and thought there was a place for gentle manipulation when possible. Cervical osteotomy was too dangerous and subtrochanteric osteotomy was to be preferred. It should be done early, after a few weeks of skin traction. Union was rapid and immobilisation was discarded at twelve weeks. *Mr T. J. Fairbank* (Cambridge) said that fixation in plaster was bad. He used internal fixation with pins and thought that growth would continue afterwards. Firm manipulation could be used but traction should be employed first. He supported the use of cervical osteotomy. *Mr E. A. Nicoll* (Mansfield) said that acute slips could always be reduced by manipulation but this must be done with great care. *Mr H. Osmond-Clarke* (London) said that Moore's pins were better than a nail but were difficult to remove. Cervical osteotomy was satisfactory if well done. *Mr W. D. Coltart* (London) emphasised the dangers of manipulation. *Mr J. C. Scott* (Oxford) advised cervical osteotomy in early cases but subtrochanteric osteotomy in late ones.

Myelography in the diagnosis of disc protrusion—*Dr Sten Cronquist* (Lund) described the use of a water-soluble contrast medium. This was irritant and it was necessary to give a lumbar anaesthetic first. The head and shoulders were kept raised for four hours afterwards and the patient was kept in bed for twenty-four hours. Any fall in blood pressure was controlled with ephedrine. The medium was absorbed in twenty minutes. Lateral, oblique and antero-posterior views were taken. Operation should not be undertaken for a few days after the examination. The findings were confirmed at operation in 92 per cent of cases. *Dr J. Mortens* (Copenhagen) said that myelography was of value in diagnosing multiple herniations but the lateral view was unreliable. Clinical signs often suggested that the lesion was higher than was in fact the case, as was made clear by myelography and confirmed at operation. Wrong centering was misleading in the interpretation of the level of the lesion. *Mr D. Lloyd Griffiths* (Manchester) said that the objection to this type of myelography in England was largely due to the dislike of a spinal anaesthetic. Fortunately, the levels at which the myelograph was of greatest value were those at which clinical diagnosis was of least value. One great advantage of oil was that a second examination was possible. Discographs from the front were of value when the anterior approach was used, and myelographs made this approach more practical. *Mr Robert Roaf* (Oswestry) emphasised the value of myelography in the diagnosis of space-occupying lesions other than simple disc protrusions, such as protrusions associated with spondylolisthesis, tumours, and occasionally diastematomyelia or traumatic paraplegia due to pressure. *Mr E. A. Nicoll* (Mansfield) strongly supported the use of myelography but said that the oil should be removed afterwards. He had found it to be 97 per cent accurate. The lumbo-sacral level was most difficult because at this level the canal widened and the theca narrowed. *Professor G. Wiberg* (Lund) said that myelographs were so good that discographs were of academic interest only. *Mr H. Osmond-Clarke* (London) said that myelography was increasingly useful but he had found headaches were sometimes troublesome. *Mr W. D. Coltart* (London) said oil should be used only when operation would follow.

Electromyography as an aid to the diagnosis of lumbar nerve compression—*Dr Bertle Knutsson* (Boraas) said that electromyography was to be preferred to, and should precede, myelography. If the findings were doubtful, operation should not be done, and after three weeks the investigation should be repeated. Electromyography was equally accurate at all levels, as well as in recurrent lesions. *Professor R. Barnes* (Glasgow) asked if double innervation confused the findings in electromyography. *Professor G. Wiberg* (Lund) replied that if enough muscles were examined it did not.

During the meeting a visit was paid to the Institution for Chronic Poliomyelitis Respiratory Paralysis where extensively paralysed patients—of whom many must rely wholly or partly on artificial

respiration—can live in their family surroundings and yet have at hand all the facilities for an emergency. Some patients spent the day with their families in flats but slept at night in the respiratory unit on the top floor. Others, less severely paralysed, might live in these surroundings but had been taught a trade at which they could work either at home or, if they are capable of it, elsewhere.

SOUTH-WEST ORTHOPAEDIC CLUB

A combined meeting of the South-West Orthopaedic Club and the Orthopaedic Section of the Royal Society of Medicine was held in Bristol on June 17, 1961, under the chairmanship of Mr A. L Eyre-Brook.

The morning was devoted to a demonstration of patients and exhibits. The afternoon was devoted to short papers each followed by discussion.

Lateral rhachotomy for vertebral biopsy in patients with a suspected tumour—*Mr N. Capener* (Exeter) described the ways in which the operation of lateral rhachotomy could be used to obtain biopsy material from the vertebral bodies in the thoracic and lumbar spines. He described the surgical technique and gave a number of illustrative examples, of which most were primary or secondary neoplasms, but one was a tuberculous lesion of the fourth lumbar vertebra which resembled a tumour. Of particular interest were two children with Calvé's disease, in both of whom the lesion was proved to be an eosinophilic granuloma. Mr Capener considered that biopsy often gave relief of pain by relieving the tension in the lesion. From this point of view it was worth while, quite apart from those patients in whom there were pressure symptoms upon the spinal cord. Needle biopsy did not have this advantage and frequently did not supply enough material for the pathologist.

Medullary nails for tibial fractures—*Mr K. H. Pridie* (Bristol) said that much experimental work had been done on this subject in Bristol and that they had the experience of treating seventy to one hundred different types of fracture by medullary nailing. A film of the technique, made by *Mr M. Alms* (Bristol), demonstrated the treatment of a recent case, the nail being introduced with the knee flexed to a right angle on a special wooden jig bolted to a standard operation table. The longest possible nail was used to fill the cortical shaft after the cancellous bone had been removed by flexible reamers. The point of insertion was on the plateau of the upper end of the tibia on the inner or outer side of the patellar tendon. The synovial cavity of the joint was not entered but the capsule was opened. Sealed underwater drainage was used for the first four hours. No splintage was used and the patient was allowed to walk as soon as the wound was healed. The period off work was about five to seven weeks. All the patients in the series maintained full ankle and subtalar movement. The difficulties of this method were described. Much discussion followed on the propriety of medullary nailing for simple fractures of the tibia. Mr Pridie pointed out that unless the method had been worked out as a routine procedure it would not have been available for difficult and complex cases where the nail saved the limb, its shape and function in a way that would be impossible by other methods.

Control of scoliosis through the growth epiphysis with particular reference to stapling by Nachlas—*Mr W. Hedley Hall* (Bath) reviewed the problem with particular regard to the theory of epiphysiodesis of the convex side of a curvature in order to correct scoliosis. He described the progress of five patients treated by vertebral stapling during the last nine years. Of these patients only one had not shown any deterioration. This was a child with hemivertebrae in whom the prognosis was in any case good. In the other four patients with idiopathic, infantile and paralytic curvatures severe deterioration had occurred despite stapling. The conclusion was that stapling had failed to control curvatures even when expected to do so. Mr Hedley Hall considered that the failure was one of method rather than one of principle, and this provoked discussion on the theories of epiphysiodesis.

Surgical treatment of the unstable cervical disc—*Mr D. M. Jones* (Bristol) discussed two aspects of the symptomatology of cervical disc lesions which sometimes led to difficulties in diagnosis. These were discomfort in the throat and localised pain in the arm, such as in the tennis elbow syndrome. He described his results in rather more than thirty patients operated on after failure to respond to conservative treatment. The operation consisted of exposure of the appropriate interval and decompression of the nerve root by removing the bone over it with a burr. No attempt was made to deal with the disc itself or to disturb the spinal cord and its coverings. The decompression was followed by a limited bone graft or, in later cases, by an acrylic insert. The patients were allowed out of bed the next day in a protective wool collar and after two weeks were discharged home in a plaster collar which remained on for three months. The results were good on the whole and several of these patients were seen during the morning's demonstration. The later progress depended to some extent on the integrity of the other cervical discs.

Spontaneous fractures of the shin in the presence of knee deformities—*Mr F. T. Wheeldon* (Plymouth) described the occurrence of spontaneous stress fractures of the tibia and fibula in three old ladies,

each of whom had angular deformities of the knee. Each had complained of aching pain in the shin for several weeks with a sudden increase of pain while walking on a level surface. The first patient was 101 years old when first seen. Her fractures united in seven weeks. She was now 104 and doing well. The second patient had flexion deformities of the knees from rheumatoid arthritis. The fractures united in seven months. The third patient also had rheumatoid arthritis and had been on steroid drugs for a long time. In her case union had not occurred. Mr Wheeldon considered that the predisposing factor in these patients was a decrease in the fatigue limit of the bone due to osteoporosis and that the flexed position of the knees predisposed them to the fractures. During discussion several surgeons described similar spontaneous fractures, the only common feature being that they all occurred in subjects with rheumatoid arthritis.

IRISH ORTHOPAEDIC CLUB

A meeting of the Irish Orthopaedic Club was held at Altnagelvin Hospital, Londonderry, on May 13, 1961. Mr N. S. Martin was in the chair.

Results of operation for lumbar disc herniation—*Mr John Lowry* (Belfast) reviewed the results of surgery in 100 cases in which the diagnosis of prolapsed intervertebral disc had been confirmed at operation. There was a relationship between the duration of symptoms before the operation and the result, patients with a short history having a much better prognosis than those with a long one. The straight leg raising test was the most reliable guide in assessing progress and in selecting patients for operation.

Influence of the flexor and adductor mechanism in arthritis of hip—*Mr A. G. O'Malley* (Liverpool) discussed the actions of the flexor and adductor muscles of the hip with special reference to the iliopsoas. He considered tightness of the latter muscle to be largely responsible for the pain and deformity in the arthritic hip. He described the release operation in which the main objective was division of the psoas tendon; a film showed the operation and some results.

The morning session ended with a demonstration of interesting cases by the host *Mr G. I. Fenton* (Londonderry). In the afternoon members toured the fine new hospital at Altnagelvin.

MEMORIAL PLAQUE TO W. J. LITTLE

On August 31 Admiral Little, grandson of William John Little and son of Muirhead Little, unveiled a plaque to the memory of the physician who founded in 1838 what is now the Royal National Orthopaedic Hospital, established tenotomy in England and described the disease named after him.

On the initiative of Dr Donald Hunter, the plaque has been placed by the London Hospital over the portal of the *Red Lion* at the junction of Aldgate and Cable Street, formerly Red Lion Street. In the old coaching inn, since rebuilt, Little, son of the landlord, was born.

The house was associated with other notable, but less reputable, characters. Dick Turpin stole a horse in Epping Forest and left it in the inn stables, where it was recognised. His accomplice, Tom King, coming to take it, was arrested. At this moment Turpin arrived and accidentally shot King.

The Little ceremony was followed by the unveiling of a similar plaque in memory of James Parkinson of the "shaking palsy" on the site of his former home in Hoxton Square by his great-great-grandson, Dr Robert Tringham.

NEW ZEALAND

NEW ZEALAND ORTHOPAEDIC ASSOCIATION

The Twelfth Annual Conference of the New Zealand Orthopaedic Association was held from June 5 to 8, 1961, at Rotorua, under the presidency of Mr Kennedy Elliot (Wellington).

Biological problems of tissue transplantation—*Professor N. W. Nisbet* (Dunedin) discussed the biological significance of the homograft reaction. Animals which had recently rejected skin homografts were in

a state of transplantation immunity, as shown by the accelerated rejection of a second skin graft from the same donor. That the transplantation antigens responsible for the homograft reaction were common to many nucleated cells was shown by the accelerated rejection of a skin homograft in an animal sensitised by bone homografts from the original donor strain. Burnet's conception of what was " self " and " not-self " remained the crux of the matter in homograft research, and his recent clonal selection theory of acquired immunity provided a useful working hypothesis. The discovery of acquired immunological tolerance by Burnet and Medawar had uncovered biological processes of evolutionary importance and not only threw light on the morphology, function and interaction of normal cells and tissues in high organisms, but also produced a conception for auto-immune disease. Experimental homograft research in which transplantation immunity and immunological tolerance were used to examine transplantation antigens, the immunological status of newborn inbred rats and on runt disease in rats (in conjunction with Dr Barbara Heslop and Dr Irmgard Zeiss) were described. *Mr Rex Blunden* (Christchurch) asked whether the induction of immunological tolerance was applicable to man by the injection of adult foreign immunologically competent cells into babies. Professor Nisbet said in reply that the appearance of runt disease in experimental animals had kept this technique entirely in the laboratory and so far was not applicable to man. It was probably dangerous to do so at present. Some reports of successful skin homografts to Rh babies treated by exsanguination and blood replacement had been reported.

Cervical spondylosis—*Mr G. J. Taine* (Napier) discussed the diagnostic difficulties of this condition and reviewed eighty-four patients. Radiographs were often not helpful. Forty-eight patients were relieved by out-patient treatment with sedation and intermittent traction, followed by restoration of neck movement by exercise; occasionally a collar was necessary. Thirty-six patients needed admission to hospital. Continuous traction was applied by means of a halter until symptoms declined and then a collar was fitted. In some patients a Minerva plaster was applied. It had not been necessary to resort to surgery. *Mr W. A. Liddell* (Christchurch) said that bizarre symptoms in cervical spondylosis might be due to associated narrowing of the vertebral or carotid arteries by arteriosclerosis which sometimes led to blackouts and falls in older people. *Mr K. Elliott* (Wellington) said that surgery had very little to offer in the treatment of this disease and he had never seen a case improved by it, but had seen some made worse. He agreed with Mr Taine that simple measures led to a high measure of relief and the importance of neurosis must be kept in mind in this condition.

Spontaneous paralysis of the posterior interosseous nerve—*Mr P. Grayson* (Palmerston North) said that this was a rare condition and he had seen only three cases. However, there was no doubt as to the etiology, and the anatomical lesion produced had been demonstrated in the cadaver and at operation. The chances were that other cases escaped notice. The lesion of the nerve was caused by excessive or unaccustomed extension of the elbow combined with supination of the forearm. This resulted in a traction or vascular lesion of the nerve where it passed from a position of relative fixation in the supinator muscle (where it was bound down by fascia) to a looser one between the superficial and deep extensor muscles of the forearm. Neurolysis of the nerves was followed by full recovery. *Mr J. Saunders* (Wellington) said that he had seen a case caused by an anatomical defect in the upper radius which had kinked the nerve. *Mr C. Milsom* (Tauranga) said that he had seen such a paralysis precipitated by unaccustomed extension of the elbow and supination of the forearm in a diabetic.

Osteotomy of the calcaneum—*Mr A. W. Beasley* (Wellington) recommended this method to correct persistent varus deformity of the heel in pes cavus. The weight-bearing position of the foot was immediately corrected and in addition the plantar structures were slackened, which reduced the clawing of the toes. *Mr P. Grayson* (Palmerston North) said that he had used calcaneal osteotomy to correct residual varus deformity of the heel in twenty-five patients between the ages of five and fifteen years with good results. *Professor N. W. Nisbet* (Dunedin) said that surgeons on the staff of the Robert Jones and Agnes Hunt Orthopaedic Hospital had used this method of correcting deformity for many years.

The disappearing hip—*Dr I. Isdale* (Rotorua, introduced by Mr C. Milsom) said that of 2,500 patients with rheumatic disease treated at the Queen Elizabeth Hospital, Rotorua, in a four-year period, radiology had revealed moderate to severe destruction of the femoral head in thirty-two hips in twenty-four patients; however, only ten of the patients in this group had received corticosteroid therapy. Dr Isdale believed that intra-articular therapy with corticosteroids, which was of the proven value, should not be discontinued in the treatment of rheumatic disease as it seemed to be impossible to predict the occurrence of this condition. *Dr B. Rose* (Rotorua) said that serological tests were useful in distinguishing between early osteoarthritic and rheumatoid hip joints. No correlation between pain, the stage of destruction of the femoral head and the dose of corticosteroids had been found. Several speakers discussed the difficulty in alleviating this condition by surgery.

Hereditary rickets—*Dr Ross Tyler* (Rotorua, introduced by Mr K. Elliot), traced affected members of one family through three generations and described the tests used to diagnose the disease, based on Professor Dent's many types of vitamin D classification. Resistant rickets was amenable to treatment with high doses of vitamin D given almost up to toxic levels. He showed a child of the family described demonstrating the radiological and clinical improvement of bow legs with this treatment. *Sir Alexander Gillies* (Wellington) said that sufficiently high doses of vitamin D had not been given in the past in this disease and in his opinion it was permissible to give near toxic doses, taking care to control them by blood phosphorus and calcium estimations. The variability of these biochemical tests must be taken into account in estimating the dose.

A clinical session, given by the staff, was held at the Queen Elizabeth Hospital, Rotorua.

Rheumatoid arthritis—*Dr B. Rose* demonstrated patients in whom the disease had begun in large joints and discussed the part played by injury in rheumatoid arthritis, which he did not think was of much importance in this respect. The treatment of rheumatoid arthritis in the Queen Elizabeth Hospital, by graduated movements and exercises, aspirin and intra-articular corticosteroids was described. After a thorough trial of conservative treatment Dr Rose said he called in the orthopaedic surgeon and had found hip arthroplasty of particular value.

Reflex patterns in cerebral palsy—*Dr B. Rose* and *Dr I. Isdale* demonstrated, with the aid of a film and patients from the cerebral palsy unit, the normal reflex movements from prematurity to the third month of life when they normally disappear. Persistence of the reflexes after the third month was a useful diagnostic aid in cerebral palsy and also helped with treatment and prognosis. Treatment was aimed at re-education of muscle groups through these reflex movements so that more normal control was gradually acquired. It was essential to start this as soon as possible, and long before the infantile patterns became established and fixed.

Fixed claw deformity of toes in rheumatoid arthritis—*Mr C. Milsom* demonstrated the post-operative condition of several patients treated by excision of the metatarsal heads through a wide transverse elliptical incision in the sole of the foot. He preferred this operation to amputation of all the toes and trimming of the under aspect of the metatarsal heads, because of the adverse psychological effects, especially in women.

Slipped upper femoral epiphysis—*Mr C. Milsom* also described the case history of a man of twenty-one years of age who had been treated six years before for bilateral slipped upper femoral epiphyses by trifin nailing of the side with slight deformity, and cervical osteotomy and trifin nailing of the side with pronounced deformity. Neither femoral head had become avascular. Following an animated discussion in which several members took part, the general opinion was that cervical femoral osteotomy was likely to be followed by a high incidence of avascular necrosis. Several speakers favoured removal of the prominent beak of the point of the femoral neck in cases with pronounced deformity and fixation of the head with Moore's pins. The trifin nail tended to displace the femoral head farther and was condemned.

Perthes' disease of the hip—*Mr C. Milsom* showed two brothers with this condition, one of whom was bilaterally affected but was symptomless, and asked whether it was justifiable to put children to bed for upwards of two years with this disease if symptoms were slight or absent and if there was no disability. Several speakers said that it was fully justified as it was the only method known to preserve the normal shape of the femoral head.

Spondylolisthesis—*Mr C. Milsom* demonstrated a patient with a moderately pronounced spondylolisthesis between the fourth and fifth lumbar vertebrae. There was no disability, but occasional backache which radiated to the buttocks. Mr Milsom believed that the etiology of spondylolisthesis was a compensatory increase of movement at the affected area due to stiffness of the spine above and below the lesion. This caused a fatigue fracture through the pars interarticularis and backward displacement of the separate neural arch, which was demonstrated in the patient. Spinal fusion of the unstable area, therefore, was not the logical treatment, and could in fact be harmful. Most cases of spondylolisthesis did not require operation, but manipulative treatment sometimes alleviated pain.

The annual golf tournament for the Gillies Cup was played at the Whakarewarewa course and was won by Mr Bruce Hay (Hamilton). At the Association dinner the president, Mr K. Elliot, described his visit to Australia as the guest of the Australian Orthopaedic Association. The election of Mr R. Blunden (Christchurch) as vice-president and president-elect of the New Zealand Orthopaedic Association was announced.

CANADA

THE DEWAR ORTHOPAEDIC CLUB

A group of Canadian orthopaedic surgeons, former residents of Dr F. P. Dewar of Toronto from 1951 to 1959, founded the Dewar Orthopaedic Club in March 1959. The purposes of the club include the advance of orthopaedic surgery by the sharing of experiences, ideas and ideals; the stimulation of academic interest in the practice of orthopaedic surgery, and the fostering of scientific investigation and presentation.

The first meeting was held in March 1959, and the second in March 1960. The last meeting was held at Kingston, Ontario, and the Lake Placid Club, New York State, in March 1961.

Meralgai paraesthetica—*Dr W. R. Ghent* (Kingston) described the variations of the lateral cutaneous nerve of the thigh at the level of the anterior superior spine and the inguinal ligament, and commented on the difficulties of diagnosis of this painful condition and the frequency of psychoneurotic symptoms. Persistent complaints could often be abolished by cutting the inguinal ligament or muscles about the anterior superior iliac spine to remove compression of the nerve. Neurectomy was seldom necessary.

Founding members of the Dewar Orthopaedic Club at Quebec. *Standing*—R. M. C. Harrison, G. A. McDonald, E. H. Simmons, G. W. D. Armstrong, J. W. Hazlett. *Sitting*—M. A. Simurda, F. P. Dewar, J. A. Leclerc. (Dr C. A. Laurin was absent on a study fellowship in Great Britain.)

Atlanto-axial derangements—*Dr W. J. S. Melvin, Dr R. F. Hetherington* and *Dr Delbert Wollin* (Kingston) discussed fractures of the odontoid process with or without dislocation, bursting fractures of the atlas, traumatic rupture of the transverse ligament of the axis, congenital anomalies, rheumatoid arthritis and recurrent rotationary subluxation of the atlanto-axial joint. They said that 30 to 40 per cent of fractures of the odontoid process unite by bone and a smaller percentage by fibrous scar. Neurological complications requiring atlanto-axial fusion were more common with subluxation than with fractures of the odontoid process.

Popliteal cysts—*Dr A. C. Derby* (Kingston) reported a series of popliteal cysts and said that there were two types. One was a true enlargement of the semimembranosus (or medial gastrocnemius) bursa, and was common in children. The second was a post-traumatic synovial protrusion of the knee joint most common in adults. The second type was often accompanied by a torn medial cartilage. The operation of medial meniscectomy and marsupialisation of the cyst from within the knee through one medial incision was described and illustrated.

Fractures in metastatic carcinoma—*Dr D. L. C. Bingham* (Kingston), outlining his experiences with this type of fracture, said that union in secondary deposits from carcinoma of the breast treated with

hormones or radiotherapy was satisfactory. Very slow union was found in fractures through deposits from carcinoma of the prostate or in multiple myelomatosis.

Fat embolism—*Dr S. L. Vandewater* (Kingston) presented the case history of an elderly patient who died of fat embolism associated with a fracture of the neck of the femur and a general anaesthetic. In the considerable discussion that followed, the consensus of opinion was that the embolic fat originated from the blood stream.

" Open sky " bone grafting—*Dr C. A. Laurin* (Montreal) reported the use of a cancellous chip bone graft to a large osteomyelitic cavity of the tibia without skin coverage. Healthy granulation tissue covered the bone chips in ten days and a split thickness skin graft, applied on the fourteenth day, was successful.

Compression in the treatment of non-union of tibial fractures—*Dr J. A. Leclerc* (Quebec City) gave several case reports in which compression, applied with pins in the tibia, had brought about union of septic ununited transverse fractures.

Arthrodesis of the wrist—*Dr D. W. Wilson* (Toronto) reviewed thirty-five patients with arthrodesis of the wrist which had been done for post-traumatic arthritis and neuropathic conditions. Twenty of the arthrodeses were of the Brittain type. Fusion usually occurred in five months but three of the Brittain grafts fractured. The best results occurred if most of the metacarpo-phalangeal joints were free of the fusion, with a graft from the radius to the capitate and base of the third metacarpal bone.

Fibroma of bone—*Dr G. A. McDonald* (Toronto) reported four patients with desmoplastic fibroma of bone, which had a destructive appearance in the radiographs, but which responded well to thorough local excision and cancellous chip bone grafting.

Proximal femoral resection for intractable hip contractures—*Dr G. W. D. Armstrong* (Ottawa) demonstrated the dramatic improvement obtained with the use of this procedure in paraplegics. A postero-lateral approach and extraperiosteal dissection were used.

Arthrodesis of joints in the growing hand—*Dr R. M. C. Harrison* (Hamilton) gave several case reports of arthrodeses obtained in finger joints between one pressure epiphysis and a metaphysis, or between two pressure epiphyses, with preservation of the epiphysial growth zones.

Synovectomy of the knee in rheumatoid arthritis—*Dr C. A. Laurin* (Montreal) reported forty-two cases of synovectomy, accompanied in twelve cases by patellectomy, for rheumatoid arthritis. The joints which had been operated upon did not become severely affected again, even with a recurrence of activity in other joints. If necessary, manipulation of the knee was most helpful, and should be done about one to two weeks after synovectomy.

Epidural injection of hydrocortisone for proven intervertebral disc protrusions—*Dr J. A. Leclerc* (Quebec City) demonstrated the technique with a film and said that sciatic pain was often relieved, at least for a few weeks or months.

Destructive lesions of the spine in Marie-Strümpell arthritis—*Dr E. A. Silverstein* (Toronto) had collected seven patients with Marie-Strümpell arthritis in whom vertebral body destruction had occurred. These lesions, which could be confused with those of pyogenic osteomyelitis, usually healed slowly with only slight kyphotic deformity.

Kissing spines—*Dr J. W. Hazlett* (Kingston) outlined the naked-eye and microscopic appearance of the lumbar interspinous areas removed at twenty necropsies. Interspinous bursae were found in thirteen of these specimens. The clinical syndrome of kissing lumbar spinous processes or interspinous bursitis was outlined and several operative specimens were shown.

GHANA

ORTHOPAEDICS AND FRACTURE SERVICES IN GHANA

Lieutenant-Colonel P. R. Wheatley, R.A.M.C., Accra, writes: In this developing country orthopaedic surgery is just beginning. There was no specialised centre until 1959 when Colonel A. B. Dempsey established an orthopaedic clinic at the civilian hospital at Korle Bu where he was given beds for women and children. At the Military Hospital, Accra, seventy men's beds were allocated for civilian injuries and orthopaedic patients. This temporary measure, pending the rebuilding of Korle Bu Hospital, is likely to last for several years. In 1960 a new children's orthopaedic ward was opened at the Military Hospital and a civilian orthopaedic surgeon, Mr Asit Mitra, was appointed. He shares the civilian work with the military surgeon.

Both Korle Bu and the Military Hospitals have up-to-date physiotherapy departments, but these are understaffed. Orthopaedic appliances are made at the Government Limb Fitting Centre. The Ghana Cripples Aid Society helps the disabled financially and seeks employment for them. These measures touch only the fringe of a vast problem, but a beginning has been made.

Traffic accidents provide the bulk of the work in the Military Hospital. The injuries are severe and often compound. Fractures of the tibia and femur predominate, but fractures of the pelvis and of the spine are also common.

In the treatment of these casualties two lessons of war surgery are forcibly brought home. Firstly, the great importance of adequate blood replacement before operation, and secondly, the value of primary debridement followed by delayed primary closure after four or five days for any compound fracture which is more than six hours old. Primary suture has been followed in several patients by gas gangrene.

Our policy in simple unstable fractures of the lower limb is open reduction and internal fixation. All suitable fractures of the uppermost third of the femur and some in the middle third are treated by intramedullary nailing. Fifty-one such operations were performed during the past twelve months without any sepsis. Patients, after nailing, are discharged from hospital on crutches within three weeks, an important factor when bed space is limited. Suitable fractures of the tibia are plated. The patients are sent out in a walking plaster; but a difficulty here is that the African will go on walking in his plaster even though it be tattered and broken, and we have had two patients with bent plates.

Bone tuberculosis is common and because of lack of beds is, in the main, treated ambulantly. Severe chronic osteomyelitis often forces a reluctant surgeon to yield a valuable bed for a sequestrectomy.

Among the children, about 60 per cent of those admitted have contractures from poliomyelitis. Almost always it is only the lower limbs that are affected and there is usually a history of an injection having been given into the buttock during the fever. Souttar's operation for contracture of the hip is the commonest operation among the children, followed closely by a gastrocnemius slide.

For the future much remains to be done. The Government are contemplating building a 400-bedded orthopaedic centre and have plans for a nation-wide programme of rehabilitation for the disabled. There is an abundance of interesting work in Ghana and no young orthopaedic surgeon would be wasting his time by spending one or two years there.

ANNOUNCEMENTS

BRITISH ORTHOPAEDIC ASSOCIATION

MEETINGS IN 1962

The spring meeting will be held in Margate from May 3 to 5. Those wishing to present a paper at the meeting should supply an abstract of about 400 words (five copies) to the Honorary Secretary, British Orthopaedic Association, 47 Lincoln's Inn Fields, London, W.C.2. The closing date for receipt of abstracts is January 3, 1962.

The autumn meeting will be held in Copenhagen. The precise dates have not been settled but it is almost certain that the meeting will be held during the last week of August and not in the first week of September as previously announced. It is important that the programme for this meeting be formulated early, and those who propose to submit a paper for consideration are requested to communicate with the Honorary Secretary, British Orthopaedic Association, 47 Lincoln's Inn Fields, London, W.C.2, as soon as possible and in any case not later than the end of January 1962.

UNIVERSITY OF LIVERPOOL

LADY JONES LECTURE

Professor Robert Judet of Paris will deliver the Lady Jones Lecture in Orthopaedic Surgery on "L'Ostéogenèse Réparatrice," on Tuesday, December 12, 1961, at 5.15 p.m., in the Large Lecture Theatre, New Medical School.

The Lecture is open to members of the medical profession, without fee or card of admission.

YUGOSLAV ASSOCIATION OF ORTHOPAEDIC AND TRAUMATOLOGICAL SURGEONS

An Association of Orthopaedic Surgery in Yugoslavia has recently been formed in Belgrade. The following officers were elected: *President*—Prof. Dr Dim Jovtchitch (Belgrade), *Vice-Presidents*—Prof. Dr F. Grospitch (Zagreb), Prof. Dr B. Brecelj (Ljubljana), *General Secretary*—Prof. Dr S. W. Stoianovitch (Belgrade), *Treasurer*—Doc. Dr C. Rakitch (Belgrade).

The first Yugoslav Orthopaedic Congress will be held in Zagreb in October 1962. The general subjects to be discussed are: "Conservative and Surgical Treatment of Congenital Dislocation of the Hip" (Lecturer—Dr F. Grospitch) and "Traffic Accidents" (Lecturer—Dr S. W. Stoianovitch). Enquiries should be directed to Prof. Dr S. W. Stoianovitch, Director of the Orthopaedic Clinic, 26 Visegradska, Belgrade, Yugoslavia.

Book Reviews

Surgical Treatment of Unequal Extremities. By Charles Weer GOFF, M.D., Associate Clinical Professor of Orthopaedic Surgery and Lecturer in Anatomy, Yale University School of Medicine; Visiting Orthopaedic Surgeon, Newington Hospital for Crippled Children. 11¼×9 in. Pp. ix+184, with 136 figures. Index. 1960. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications Ltd. Price 88s.

This monograph is concerned not only with the technique in the surgical treatment of inequalities of the lower limbs, but deals also with factors influencing bone growth and kindred matters. The author enumerates and examines the various methods of measuring long bones and bone growth, and of predicting its probable extent. While favouring certain procedures, he states that " whatever percentage is selected for predictional values, there is an error in measuring long bones by any method." The various schemes that have been advocated during the last century for stimulating the growth of long bones—from venous stasis through juxta-epiphysial drilling, the introduction of foreign bodies such as ivory pegs and dissimilar screws, divers osteotomies to lumbar sympathectomy—are weighed in the balance, and, on the whole, found wanting. The temporary arrest of epiphysial growth by stapling is set out in some detail, and the permanent epiphysiodesis of Phemister and others described. A chapter is devoted to a study of the human epiphysis before and after stapling. It contains many excellent micrographs, and is a valuable contribution.

The chapter on femoral, tibial and fibular lengthening and shortening is less satisfactory. Few surgeons will disagree with the author's contention that the best method of shortening a femur is by an oblique sliding osteotomy of the shaft sustained at the desired length by two or more screws, nor that the tibia and fibula can be shortened satisfactorily, but there is justification for non-acceptance of his unequivocal condemnation of leg lengthening. On this subject illustrations of Abbot's original apparatus (1927) are produced. These have as little resemblance to modern equipment as the quite unnecessary and possibly mutilating exposure, that the author deems to have been established on operative principles, has to modern methods, where the exposure is reduced to the minimum. The author expresses his happiness that the days of leg lengthening are over at Newington, where apparently five cases were done. The records of two of these are lost, and osteomyelitis developed in at least one of the remaining three. In other hands leg lengthening has proved a very useful procedure, especially in cases of poliomyelitis, but not practised along the lines the author describes.

The monograph is well produced, with copious illustrations, graphs and tables. It has a very useful bibliography, and should be read by all who are interested in the subject.—Robert I. STIRLING.

Atlas of Anatomy and Surgical Approaches in Orthopaedic Surgery. Volume Two—Lower Extremity. By Rodolfo COSENTINO, M.D., Assistant Professor in Orthopaedic Surgery, University of La Plata, Argentina; Research Associate, Department of Orthopaedic Surgery, State University of Iowa, Iowa City. With a Preface by Carroll B. Larson, M.D., Professor and Head, Department of Orthopaedic Surgery, State University of Iowa. 11¼×9 in. Pp. xii+264, with 139 figures. 1960. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications Ltd. Toronto: The Ryerson Press. Price £5, 12s.

Last year I was privileged to review the *Atlas of Anatomy of the Upper Extremity*. Now Dr Cosentino has completed his volume on the lower extremity. This is anatomy for the operating orthopaedic surgeon and it will be of great help to the surgeon in training before he carries out standard operations which to him are new procedures. It will also be helpful to the more experienced surgeon who is to undertake the exploration of anatomically complex and perhaps unfamiliar areas. The photographs of dissections present the surgical anatomy with great clarity. Again I can commend this work.—J. I. P. JAMES.

Intra-osseous Venography. By Robert A. SCHOBINGER, M.D., Clinical Instructor in Surgery, Albert Einstein College of Medicine, New York, N.Y.; Section Chief, Surgical Service, Veterans Administration Hospital, Bronx, N.Y.; formerly Associated Cancer Research Surgeon, Roswell Park Memorial Institute, Buffalo, N.Y. With a foreword by Philip J. HODES, M.D. 10½×7 in. Pp. xii+243, with 317 figures. Index. 1960. New York and London: Grune & Stratton, Inc. Price $14.50.

Intra-osseous injection of contrast medium can outline radiographically the internal structure of bone and reveal the various venous pathways that drain the area concerned. This treatise, clearly written by an expert, is devoted to the various applications of this diagnostic method. The work begins with a consideration of the technical aspects of intra-osseous venography and this is followed by a section on osteomyelography in which venous patterns within bone are discussed. The remaining eleven chapters are devoted to the radiology of the various major venous pathways that can be demonstrated by intra-osseous injection. Each chapter starts with a section on the local venous anatomy, then describes the positioning of the patient and injection technique, and ends with a discussion on the clinical application of the method. All the regional chapters are illustrated by a series of venograms. These have not rendered good reproductions, but a clear line drawing accompanies each radiograph and interpretation is not difficult. The azygos system of veins lends itself well to study by intra-osseous injection and one of the major chapters is concerned with "Azygography." In this chapter the author's concept that altered haemodynamics can produce characteristic changes in the venogram is well illustrated by the "azygographic" pattern in portal hypertension. Similarly, another long section deals with venography of the vertebral venous plexus and is illustrated by venograms in such diverse conditions as herniated intervertebral disc, spondylolisthesis and vertebral metastases.

The author is strongest when dealing with the purely technical aspects of the method and is less sure when discussing the indications for intra-osseous venography. He has established, however, that intra-osseous venography should have a place in the armamentarium of the modern x-ray diagnostician and this book can be recommended as a practical guide to this highly specialised technique.—G. W. TAYLOR.

Verletzungen und Wiederherstellung der Oberen Extremitäten Einschliesslich der Hand. By V. STRUPPLER and N. WITT. Erster Teil—**Die Frischen Verletzungen.** By Prof. Dr Victor STRUPPLER, Chirurgische Poliklinik der Universität München. 10¼×7½ in. Pp. xii+211, with 663 illustrations in 178 figures, some in colour. Index. 1961. Neue Deutsche Chirurgie, Band 68. Stuttgart: Ferdinand Enke Verlag. Price DM 67.

This short volume is best described to English readers as a combination of Rank and Wakefield on *Surgery and Repair as Applied to Hand Injuries* and Watson-Jones on injuries to the upper extremity; indeed a good many familiar illustrations from the latter will be met with in its pages. It is fully and magnificently illustrated in black and white and in colour. It follows accepted precepts and the only general view it purveys which is susceptible to criticism is a slight over-emphasis on mechanical fixation in complicated fractures about the elbow. In contrast to this the reduction of the dislocated acromio-clavicular joint by a single screw in the coracoid process is omitted in favour of Bunnell's fixation by a fascial strip. As befits their importance, half the volume is devoted to injuries to the hand. To include all injuries to the upper extremity in the remaining half necessarily entails a rather abbreviated treatment; for instance, posterior dislocation of the shoulder is mentioned in one line as a rarity. It is an admirable text-book for the senior registrar and it is a pity its attractive conciseness is marred by an inadequate index.—J. G. BONNIN.

Pain. Problems Pertaining to the Transmission of Nerve Impulses which give rise to Pain. Preliminary Statement. By W. NOORDENBOS, Surgeon-in-Charge, University Neurosurgical Department, Wilhelmina-Gasthuis, Amsterdam. 9×6 in. Pp. 182, with 40 figures. Index. 1959. Amsterdam, London, New York, Princeton: Elsevier Publishing Company. Distributors for Elsevier: D. Van Nostrand Company Ltd. Price 42s. 6d.

This monograph is based upon an attempt to find common features in patients suffering from pain of uncertain origin such as post-herpetic neuralgia, atypical facial pain, phantom limb, causalgia, etc. The author stresses the great difficulty of describing disagreeable sensations in words and of assessing the severity of chronic pain.

The physiology of the conduction of pain impulses is discussed and the literature on the subject is widely reviewed. Dr Noordenbos believes that the persistence of painful sensations after neurological

injuries is due to the existence of a multi-synaptic afferent system within the spinal cord. This, he suggests, is activated and inhibited by impulses reaching it from the periphery; if the balance of some impulses is disturbed by injury, long-lasting painful sensations may result. In the same way he explains why operations designed to denervate the painful areas may fail to relieve the condition.

The book will be of interest and value to those seeking an explanation for persistent pain of obscure origin, but will assist little in the practical alleviation of pain.—Campbell CONNOLLY.

Instructional Course Lectures. Volume XVII. The American Academy of Orthopaedic Surgeons. Edited by Fred C. REYNOLDS, M.D., St Louis, Missouri. 11×9 in. Pp. 421, with 358 figures and 28 tables. Index. 1960. St Louis: The C. V. Mosby Company. London: Henry Kimpton. Price £6, 18s.

This is the seventeenth volume of Instructional Course Lectures and is a selection of the lectures given at the 1960 meeting of the American Academy of Orthopaedic Surgeons. It is unquestionably the best volume that has appeared so far, and your reviewer has read most of it with considerable pleasure and profit. The outstanding article is by Blount on "Unequal Leg Length and its Management"; but there are many other good things—Green and Anderson on "Skeletal Age and the Control of Bone Growth," a section on bone graft surgery which includes a superb review by Urist of the research in this subject in the past ten years, and two practical articles on "The Principles of Bone Graft Surgery" by Herndon and "The Causes and Treatment of Non-Union" by Boyd and Lipinski. There are also valuable contributions by Robinson and Southwick on "Surgical Approaches to the Cervical Spine," Fahey on "Fractures of the Elbow in Children" and Stokes on "Surgical Management of Vascular Injuries Associated with Long Bone Fracture."

In 1953 James Dickson named the subcapital fracture of the neck of the femur "the unsolved fracture." No one reading the group of articles on this subject will doubt the aptness of the term, because almost all the contributors have described a different method of internal fixation, and there is surprisingly little agreement on the causes of failure. Any orthopaedic surgeon who thinks he knows the answers should read this section; those who, like myself, are in search of facts rather than opinions may be disappointed.

The book is well produced, and the quality of the illustrations is better than in some of the earlier volumes, but there is still room for improvement. One suspects that some of the radiographs submitted are neither suitable for reproduction nor essential to the understanding of the text. Would it not be better to exclude them and pass on the benefit to the reader?—Roland BARNES.

Cancer Progress Volume 1960. Edited by Ronald W. RAVEN, *O.B.E.(Mil.)*, *T.D.*, F.R.C.S., Joint Lecturer in Surgery, Westminster Medical School, University of London; Surgeon, Westminster Hospital Teaching Group; Surgeon, the Royal Marsden Hospital, London; Surgeon, the French Hospital, London; with twelve advisory editors and thirty-three other contributors. 10×7 in. Pp. xvi+258, with 45 figures and some tables. Index. 1960. London: Butterworths. Price 60s.

Cancer Progress is a companion volume to the larger work on cancer and is a very readable and stimulating account of current cancer research. Whilst the volume may contain little of direct interest to orthopaedic surgeons, it is important that we should be aware of the exciting developments that are occurring in many fields of cancer surgery, and be ready to introduce the recent diagnostic and therapeutic techniques into the management of malignant tumours of bone and soft tissue. All malignant tumours of bone have a high mortality rate, none more so than osteosarcoma. The problem here is a rapid dissemination of the tumour often within a few weeks of the first symptoms. In this context the account of circulating cancer cells in the blood stream by Stewart is of great interest. Is biopsy as harmless a procedure as is often supposed? A count of the sarcoma cells in the venous blood draining a limb before and after biopsy might be informative.

There are articles on the chemotherapy of cancer and regional perfusion with chemotherapeutic agents. Stehlin and Clark, the originators of limb perfusion, make no exaggerated claims for the method. They state firmly that "as currently employed its long term therapeutic value may prove to be insignificant, hence it is not recommended as a standard weapon in the treatment of patients with cancer." It would appear that the most useful field for the cytotoxic drugs "may be the inactivation of circulating cancer cells by chemotherapeutic compounds, whilst the primary tumour is removed surgically or destroyed by radiotherapy." Until a cytotoxic agent is developed which has a greater affinity for the cancer cells, it may be necessary to counteract the damage to the haemopoietic tissues

by bone marrow replacement, a subject discussed in an authoritative fashion in two articles by Kay and Koller, and Thompson Hancock.

Strickland gives an account of the place of arteriography in the diagnosis of tumours of bone and soft tissue, and there is a valuable paper on the oxygen effect in radiotherapy by Churchill-Davidson, and a fascinating report on cancer in the South African Bantu by Oettlé.

Your reviewer recommends this book enthusiastically to anyone interested in the cancer problem. —Roland BARNES.

Disc Lesions and Other Intervertebral Derangements. Treated by Manipulation, Traction and other Conservative Methods. By E. J. CRISP, M.D.(Cantab.), D.Phys.Med.(Lond.), Consultant Physician Emeritus in Physical Medicine, Guy's Hospital, London. 8½×6 in. Pp. viii+158, with 48 figures. Index. 1960. Edinburgh and London: E. & S. Livingstone Ltd. Price 15s.

The preface suggests that this book will be mainly concerned with the value of manipulation in disorders of the spine. This is far from the case; neither is the author concerned especially with lesions of the intervertebral discs, but takes great pains to describe other minor vertebral disorders which can confuse the diagnosis of backache. It cannot truly be said that Crisp does much to clarify a situation in which diagnosis remains intuitive and so often based on theories of pathology which have not been confirmed by surgical experience or post-mortem findings. Crisp is opposed to manipulation of the spine in acute low back pain, but rather tentatively suggests that manual traction may succeed when bed rest has not been rapidly effective. The place of manipulation is in the treatment of chronic low back pain when serious disorder has been ruled out by radiography and other investigations. He is not concerned entirely with the problem of cervical or lumbar backache, and there is a chapter on the thoracic (dorsal) region, in which manipulation is used for the relief of " intercostal neuritis " due to some causes other than acute nuclear prolapse, widespread spondylosis, Scheuermann's disease, vertebral and spinal tumours, or herpes zoster; this type of intercostal pain he ascribes mostly to " costo-vertebral " derangements.

This small book, which can be read quickly, ends with a section on the basic techniques of manipulation and traction.—W. D. COLTART.

Surgery is Destined to the Practice of Medicine. Hunterian Oration Royal College of Surgeons of England. By Sir Reginald WATSON-JONES, B.Sc., M.Ch.Orth., F.R.C.S., F.R.A.C.S.(Hon.), F.R.C.S.E.(Hon.), F.A.C.S.(Hon.), Orthopaedic Surgeon-Extra to Her Majesty the Queen, Orthopaedic Surgeon to His late Majesty King George VI. 10×7 in. Pp. 81, with 77 figures (many in colour). 1961. Edinburgh and London: E. & S. Livingstone Ltd. Price 21s.

On February 16, 1959, Sir Reginald Watson-Jones had the honour of delivering the eighty-ninth Hunterian Oration at the Royal College of Surgeons of England, before a large and distinguished audience. He took as his text a quotation from John Hunter—" This last part of surgery, namely operations, is a reflection on the Healing Art, it is a tacit acknowledgment of the insufficiency of surgery." He claimed that the truth of this statement is demonstrated by the history of orthopaedic surgery over the past fifty years, during which time the gross deformities caused by tuberculosis, poliomyelitis and other diseases have virtually disappeared as a result of preventive medicine and of the greatly increased knowledge of pathological conditions in general. Those who were fortunate enough to hear Sir Reginald on that memorable occasion realised that he took his place amongst the great Hunterian orators. I strongly recommend this little volume, for to read it is a delight.—F. W. HOLDSWORTH.

Geschichte der Orthopädie. By Prof. Dr Bruno VALENTIN, Rio de Janeiro. 9¾×7 in. Pp. xvi+288, with 142 figures. Index. 1961. Stuttgart: Georg Thieme Verlag. Price DM 39.60.

As the author points out, it is indeed surprising that no general history of the development of the art of orthopaedics had yet been written, and Professor Valentin of Rio de Janeiro has done us all a great service in undertaking this task. He has travelled far and wide in his search for the best material and sources of information, and consequently has produced a scholarly book of reasonable proportions which will be gratefully received throughout the world. After brief reference to orthopaedics in the pre-Christian civilisations, he gives us in the first half a survey of the development of understanding and methods of treatment in the various major orthopaedic diseases; this is the best part of the book. In the second part he deals with the history of orthopaedic surgery country by country throughout

Europe and America. No doubt there will be criticism from some nations that their contribution has received insufficient space or recognition, but within the compass of a book of this size a certain degree of superficiality must be accepted. We should all be grateful to the author for undertaking this important work, doing it with balance and judgment, and producing such a well-illustrated and eclectic contribution to medical history.—T. J. FAIRBANK.

Key and Conwell's Management of Fractures, Dislocations, and Sprains. By H. Earle CONWELL, M.D., F.A.C.S., Associate Professor of Orthopedic Surgery, University of Alabama School of Medicine, Birmingham, Alabama; Attending Orthopedic Surgeon, University Hospital, St Vincent's Hospital, Children's Hospital, Baptist Hospitals, East End Hospital, and South Highlands Infirmary, Birmingham, Alabama; Consulting Orthopedic Surgeon, Veterans Hospitals, Tuscaloosa, Alabama, and Montgomery, Alabama; Member, Trauma Committee, American College of Surgeons; Member, Fracture Committee, American Academy of Orthopaedic Surgeons; and Fred C. REYNOLDS, M.D., Professor of Orthopedic Surgery, Washington University School of Medicine, St Louis, Mo. Seventh edition. 10×8 in. Pp. 1,153, with 1,227 figures. Index. 1961. St Louis: The C. V. Mosby Company. London: Henry Kimpton. Price £10, 2s. 6d.

This well known text-book reaches its seventh edition. Each time I review it, and this is the third, I find numerous improvements. I still believe that both text and diagrams could be simplified and reduced with increased clarity, although this is considerably improved. The book is of course concerned with American practice. A most thoughtful and conservative approach to the treatment of fractures includes a sound appreciation of the value and dangers of intramedullary fixation. The section on fractures in the hand is much improved and is now excellent. After seven editions this book scarcely needs commendation. It certainly deserves it.—J. I. P. JAMES.

The Closed Treatment of Common Fractures. By John CHARNLEY, B.Sc., M.B., F.R.C.S., Orthopaedic Surgeon, Manchester Royal Infirmary; Orthopaedic Surgeon, The Park Hospital, Davyhulme; Orthopaedic Surgeon, Wrightington Hospital; Honorary Lecturer in Orthopaedics, Manchester University. Third edition. 10×7½ in. Pp. xii+272, with 215 figures, some in colour. Index. 1961. Edinburgh and London: E. & S. Livingstone Ltd. Price 50s.

Mr Charnley has honoured us by preparing a third edition of *The Closed Treatment of Common Fractures*. He is one of the most interesting, stimulating and enigmatic figures in British orthopaedic surgery. Perhaps it is because he is enigmatic that he is so stimulating. At one moment you find yourself in complete agreement with his sound teaching and within seconds are infuriated by his observation. This is the basis of good teaching. You are never allowed to go to sleep, changing only from warm approval when you want to clap aloud, to stern disapproval when you just frown. This is the calibre of his brilliant monograph which every orthopaedic surgeon should read, study and have in his library. I trust very much that they will regard as nonsense the idea that displacement of fractured bones should be increased before they can be reduced. Figure 105 on page 131, showing reduction of a Colles fracture, fills me with horror, and I am glad only that he does not now emphasise the same idea in treating supracondylar fractures of the humerus. But such criticism confirms the success of this stimulating monograph which is so rewarding and so exciting that every young surgeon should read it again and again.—Reginald WATSON-JONES.

Pediatric X-ray Diagnosis. By John CAFFEY, A.B., M.D., Director of Medical Education, Children's Hospital of Denver; Consultant Pediatrician, National Jewish Hospital at Denver, Colorado; Professor Emeritus of Radiology, College of Physicians and Surgeons, Columbia University; Consultant Radiologist, Columbia-Presbyterian Medical Center, New York City. Fourth edition. 10½×8½ in. Pp. xxvi+1,236, with 1,536 figures. Index. 1961. Chicago: Year Book Medical Publishers, Inc. Distributed in the United Kingdom by Interscience Publishers Ltd. Price 240s.

This excellent text-book, first published in 1945, is now in its fourth edition. It stems from the Roentgen Conference held every fortnight in the Babies' Hospital, New York. The purpose of the author is two-fold: description of shadows cast by normal and morbid tissues, and clinical appraisal of roentgen findings in pediatric diagnosis. Roentgen physics, technique and therapy have been intentionally omitted.

The most pressing current problem in pediatric radiology is the reduction of hazards to the patient from exposure to ionising radiation, because these patients are the most vulnerable of all human beings. Roentgen examinations should be made when there are good clinical indications, and the only good clinical indication is a reasonable probability that the individual patient will benefit personally. It is good to have this emphasised and the author rightly condemns repetitious, elaborate techniques, repeated examinations and fluoroscopy, the last being the greatest source of heavier exposures. One thinks particularly of the far too frequent exposure to the rays of the little girl with congenital dislocation of the hip, often—in fact usually—without adequate protection with lead guards to shield the ovaries. Methods of reducing exposure are described.

Pediatric radiology continues to grow so that its body of knowledge is already too great for anyone to master and consequently branching and subdivisions of this supposed speciality are beginning to appear. In this edition 127 new and important items are added. Some of the more important include eleven new variants in the skull, cranial changes in iron-deficiency anaemia, drawings of the maturation of the normal cervical spine, eventration of the diaphragm in infantile cortical hyperostosis, the pelvis in mongolism, multiple coronal cleft vertebrae, and a new section on rheumatoid arthritis.

It is obvious that although all child's disease is included, orthopaedic pathology is responsible for a large part of the volume. A somewhat rare condition, Pyle's disease or idiopathic symmetrical splaying of the long bones, is being recognised with increasing frequency. It is well described and illustrated. It is interesting to see complete restoration of a vertebra in vertebra plana (Calvé) in a period of twenty-two years. There is increasing doubt that patients who exhibit the signs of Osgood-Schlatter's condition have necrosis of the tibial tubercle, traumatic avulsion and fragmentation being more likely. All the " normal variations " in the size and configuration of the tubercle are depicted, as are variations in many parts of the skeleton. Infantile cortical hyperostosis may persist in a chronic condition for years and leave crippling deformities. There are some good illustrations of the condition. So, too, are there of the various forms of avitaminosis. The resemblance of the lesions in hypophosphatasia to rickets is noted. The section on skeletal changes in the endocrinopathies is interesting, well described and illustrated, including oxalosis, idiopathic hypercalcaemia and Cockayne's syndrome.

Curiously enough only a few pages are devoted to the study of congenital dislocation of the hip. One would like to have seen more illustrations of arthrographs accompanied by descriptions, for these arthrographs are very difficult to read.

This is a large volume, as it must be. It contains much of great interest to the orthopaedic surgeon and in the reviewer's opinion is a " must " for those practising this speciality. The letterpress is good and the radiographs, although indifferently reproduced, are clear and adequate.—Walter MERCER.

Varicose Veins. A Practical Manual. By R. Rowden FOOTE, M.R.C.S., L.R.C.P., London. Third edition. With the assistance of A. Gordon DINGLEY, M.A., M.Chir.(Cantab.), F.R.C.S., Consultant Surgeon to the Southend-on-Sea Group of Hospitals; Late Surgical Chief Assistant, St Bartholomew's Hospital, London. 10×7 in. Pp. xx+356, with 304 figures, some in colour. Index. 1960. Bristol: John Wright & Sons Limited. Price 63s.

Few orthopaedic surgeons have taken a serious interest in the disorders of the veins of the lower limbs. With the third edition of his book Mr Rowden Foote provides an agreeable opportunity for repairing this omission. At once comprehensive and readable, this book well combines discussion of theoretical considerations and experimental results with practical instruction in methods of diagnosis and treatment. Logical arrangement of chapters and excellent illustration are well supplemented by an extensive bibliography and by accurate indices. There is an occasional irregularity in the writing, but the intention always remains clear.

Since 1954, when the second edition of *Varicose Veins* appeared, there have been adjustments of thought in several important fields of work on veins. In particular, further evidence has been produced on the value of the stripping operation and on the effect of treatment with anticoagulants, and the relation of deep venous thrombosis to ulceration has been further explored. These and other new aspects have been covered by rewriting and by additions, and it is pleasant to find that a scrupulous impartiality has been maintained throughout these reappraisals.

It is twelve years since Mr Rowden Foote first produced his practical manual for the surgeon specially interested in the treatment of varicose veins. He and Mr Gordon Dingley now go further to show us this special field as one in which much progress has been made, and which holds much of importance to all concerned with the surgery of the lower limb.—George BONNEY.

BOOKS RECEIVED

These books are acknowledged with grateful thanks. Selections will be made for review as space permits and in accordance with their interest for our readers.

Surgery of Trauma in the Tropics. By R. L. BATTEN, B.A.(Cantab.), F.R.C.S. 1961. London: Edward Arnold (Publishers) Ltd.

Die Spondylosis Cervicalis. By Privatdozent Dr med. Max AUFDERMAUR. 1960. Stuttgart: Hippokrates-Verlag.

Bone Homografts in the Surgical Treatment of Cysts of the Jaws and Periodontal Pockets. By Heyman KRØMER. 1960. Oslo: Oslo University Press.

Cancer—The Significance of Delay. By Robert SUTHERLAND, M.D., D.P.H. 1960. London: Butterworth & Co. (Publishers) Ltd.

L'Echinococcosi Vertebrale. By Antonio CABITZA. 1960. Roma: Il Pensiero Scientifico Editore.

Ervaringen met Spondylodese bij de Behandeling van Lumbosacrale Insufficientie. By J. A. VAN DUREN. 1960. Leeuwarden: Drukkerij Westerbaan.

Congenital Deformities. By Gavin C. GORDON, M.B., F.R.C.S.E. 1961. Edinburgh and London: E. & S. Livingstone Ltd.

Roentgenographic Studies on Epiphysial Growth and Ossification in the Knee. By Sven SCHELLER. 1960. Stockholm: Acta Radiologica, Supplementum 195.

Leprosy: Its Challenge and Hope. By R. G. COCHRANE, M.D., F.R.C.P., D.T.M.& H. 1960. London: The Mission to Lepers.

Le Coude en Pratique Rhumatologique. By S. DE SÈZE and M. MAITRE. 1960. Paris: Masson & Cie, Editeurs.

Neuroradiology Workshop. Volume I: Scalp, Skull and Meninges. By Leo M. DAVIDOFF, M.D., Harold G. JACOBSON, M.D., and Harry M. ZIMMERMAN, M.D. 1961. New York and London: Grune & Stratton, Inc.

Die Behandlung der Schädelbasisbrüche. By Prof. Dr H.-G. BOENNINGHAUS. 1960. Stuttgart: Georg Thieme Verlag.

Artefacts and Handling and Processing Faults on X-ray Films. By Prof. Dr E. A. ZIMMER. 1960. New York and London: Grune & Stratton.

Les Tractions Vertébrales. By J. LEVERNIEUX. 1961. Paris: Expansion Scientifique Francaise.

Cerebral Palsy in Childhood and Adolescence. Edited by J. L. HENDERSON, M.D., F.R.C.P.E. 1961. Edinburgh and London: E. & S. Livingstone Ltd.

Pathology of the Nervous System. By J. Henry BIGGART, *C.B.E.*, M.D., D.Sc., F.R.C.P. Third edition. 1961. Edinburgh and London: E. & S. Livingstone Ltd.

Die Myelographie mit Positiven Kontrastmitteln. By Dr J. WELLAUER. 1961. Stuttgart: Georg Thieme Verlag.

Roentgen Diagnosis in Orthopaedic Traumatology. By S. L. WEISSMAN, M.D. 1960. Tel-Hashomer Government Hospital, Israel: The Office of the Surgeon General.

Theory of Shoulder Mechanism: Descriptive and Applied. By A. K. SAHA, B.Sc., M.B., B.S., F.R.C.S., F.R.C.S.Ed., M.Ch.Orth. 1961. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications Ltd.

La Radiculographie Lombo-sacree par Substance Iodée Hydrosoluble et Résorbable. By Jacques FERRAND, Jean ROBERT D'ESHOUGUES and Jacques BARSOTTI. 1961. Paris: Expansion Scientifique Francaise.

Mineral Metabolism. Edited by C. L. COMAR and Felix BRONNER. Volume I. Principles, Processes, and Systems. Part B. 1961. New York and London: Academic Press, Inc., Publishers.

Synovial Joints. Their Structure and Mechanics. By C. H. BARNETT, M.A., M.B., F.R.C.S.; D. V. DAVIES, M.A., M.B., B.S.; and M. A. MACCONAILL, M.B., D.Sc., M.R.I.A. 1961. London: Longmans, Green and Co. Ltd.

Pseudarthrosis of the Long Bones. By Otto VERBEEK, M.D., and C. P. DUBBELMAN, M.D. 1961. Amsterdam: North-Holland Publishing Company.

Medical Evidence in Personal Injury Cases. By Dorothy KNIGHT DIX and Alan H. TODD, M.S., B.Sc.Lond., F.R.C.S. 1961. London: H. K. Lewis & Co. Ltd.

The Spinal Cord. By George AUSTIN. 1961. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications Ltd.

ACKNOWLEDGMENTS

We acknowledge with thanks receipt of:

Acta Chirurgica Scandinavica, and Supplements
Acta Orthopaedica Italica
Acta Ortopedica-Traumatologica Iberica
Annals of the Royal College of Surgeons of England
Archives of Surgery
Biological Abstracts
Boletin de la Sociedad de Cirugia del Uruguay
British Journal of Surgery
British Medical Bulletin
Canadian Journal of Surgery
Indian Journal of Surgery
Journal of Neurology, Neurosurgery and Psychiatry
Journal of the American Medical Association
Journal of the Royal College of Surgeons of Edinburgh
Lancet
Ortopedia e Traumatologia dell'Apparato Motore
Radiology
Revista de Ortopedia y Traumatologia
Revue de Chirurgie Orthopédique
Surgery, Gynecology and Obstetrics
Zeitschrift für Orthopädie und ihre Grenzgebiete

AUTHOR INDEX

A

Abercrombie, G. F. Major injuries (*symposium*), 182.
Adorjan, S. Severe slipping of upper femoral epiphysis, 405.
Allcock, E. A. Torulosis, *71.
Alldred, A. Congenital absence of radius (*discussion*), 189.
Allen, C. E. L. Intervertebral disc excision and lumbar spine fusion by a transperitoneal abdominal approach (*discussion*), 401.
Occult forms of spinal dysraphism (*discussion*), 604.
University of Cape Town artificial limb, 403.
Alms, M. Fracture mechanics, *162.
See also Pridie, K. H., and Alms, M.
Alvik, I. Congenital dysplasia of hip: clinical review, 193.
Arden, G. P. *See* Maudsley, R. H., and Arden, G. P.
Armstrong, G. W. D. One-stage pantalar arthrodesis, 610.
Proximal femoral resection for intractable hip contractures, 863.
Aston, J. N. *Book reviews*, 413, 419.
Aubigné, R. Merle d'. *See* Merle d'Aubigné, R.

B

Bachynski, B., and Spooner, H. J. Hyperthyroidism in polyostotic fibrous dysplasia, 613.
Badgely, C. Femoral anteversion (*discussion*), 603.
Treatment of displaced subcapital fractures of femoral neck in the aged by immediate replacement arthroplasty (*discussion*), 606.
Bado, J. L. Etiology and treatment of congenital dysplasia of hip, 196.
Baker, L. D. Indications for early surgery in cerebral palsy, 203.
Ball, J. Cellular immunity and the bone graft (*discussion*), 601.
Mobility and instability in the cervical spine, 600.
Barber, J. R. *See* Haliburton, R. A., Brockenshire, F. A., and Barber, J. R.
Barlow, T. G. Congenital dislocation of hip, 603.
Barnes, R. *Book reviews*, 867.
Electromyography as aid to diagnosis of lumbar nerve compression (*discussion*), 857.
Multiple injuries mainly involving lower limbs (*discussion*), 606.
Paraplegia in extension injuries of spine (*discussion*), 600.
Surgery of primary osteoarthritis of hip (*discussion*), 857.
Barnett, E. *See* Stevens, J., *et al.*
Barrett, A. M. *See* Fairbank, T. J., and Barrett, A. M.
Batchelor, J. S. Femoral anteversion (*discussion*), 603.
Bauer, G. C. H. Accident services (*discussion*), 854.
Epidemiology of fracture in old persons, 201.
Osteoporosis, 856.
Surgery of primary osteoarthritis of hip (*discussion*), 856.
Bazileuskaya, Z. Spinal fusion for scoliosis, 204.
Beare, R. L. B. *Book review*, 415.
Beasley, A. W. Osteotomy of calcaneum, 860.
Bell, H. *Discussions*, 401.
Fibular by-pass operation for non-union of tibia, 404.
University of Cape Town artificial limb (*discussion*), 403.
Berk, M. E., and Tabatznik, B. Cervical kyphosis from posterior hemivertebrae, *77.
Berry, R. J. Genetically controlled degeneration of the nucleus pulposus in the mouse, *387.
Bertelsen, A. Accident services (*discussion*), 854.
Slipped upper femoral epiphysis, 857.
Bertrand, P. Congenital dysplasia of hip (*discussion*), 197.
Besa, A. S. Use of iliac crest as full-thickness graft in spinal fusion, 202.
Betts, W. J. Condylar fractures of elbow, 614.
Bickerton, G. Fractures of spine and their treatment (*discussion*), 401.
Bigley, D. Minor injuries: anaesthesia and analgesia, 183.
Billewicz, W. Z. *See* Mitchell, P. E. G., Hendry, N. G. C., and Billewicz, W. Z.
Billing, L. Slipped upper femoral epiphysis studied by a new radiographic technique, 187.
Bingham, D. L. C. Fractures in metastatic carcinoma, 862.
Blackwood, H. J. J. Anatomy and pathology of mandibular joint, 396.
Blockey, N. J., and Schorstein, J. Intraspinal epidermoid tumours in lumbar region of children, *556.
Blount, W. P. Congenital scoliosis, 202.
Blunden, R. Biological problems of tissue transplantation (*discussion*), **860**.
Böhler, L. Organisation of hospital services, 201.
Boerema, I., and Brummelkamp, W. H. Treatment of anaerobic infections with oxygen under pressure, 407.
Bolton, H. Multiple injuries mainly involving lower limbs (*discussion*), 605.
Treatment of displaced subcapital fractures of femoral neck in the aged by immediate replacement arthroplasty, 606.
Bonilla, A., and Guerra, G. Congenital luxation of hip, 194.
Bonney, G. L. W. *Book review*, 870.
Bonnin, J. G. *Book reviews*, 210, 409, 417, 418, 866.
Bosc, F. V. A. Major injuries: rehabilitation, 182.
Bowden, R. E. M., and Napier, J. R. Assessment of hand function after peripheral nerve injuries, *481.
Boyd, H. B. The study of osteogenesis with its relation to delayed union and pseudarthrosis (*symposium*), 197.
Boyd-Wilson, J. S. Diagnosis of lumbar disc degeneration, 190.
Braddock, G. T. F. Prolonged follow-up of peroneal spastic flat foot, *734.
Brand, P. W. Tendon grafting, *444.
Brecelj, B. Radiographic observations in the course of post-natal spontaneous evolution of dysplastic hips, 196.
Bremner, E. *See* Valls, J., Bremner, E., and Sequers, A. M.
Brett, M. S. Influence of certain hip muscles upon shape of upper end of femur (*discussion*), 174.
Brimblecombe, F. Early diagnosis of cerebral palsy, 398.
Bro-Rasmussen, F. Embryological development with special reference to congenital deformity, 405.
and Hansen, O. M. Striated muscle tissue, 405.
Brockenshire, F. A. *See* Haliburton, R. A., Brockenshire, F. A., and Barber, J. R.
Brook, A. L. Eyre-. *See* Eyre-Brook, A. L.
Brookes, M. *See* Wardle, E. N., and Brookes, M.
Bruce, J. *McMurray Memorial Lecture*, 395.
Brummelkamp, W. H. *See* Boerema, I., and Brummelkamp, W. H.

* Indicates full article.

Buck, J. E. Recurrent dislocation of shoulder (*discussion*), 184.
Bukh, N. Muscle regeneration: incidence of scoliosis and foot deformities after poliomyelitis, 853.
Bull, J. Burns: treatment in hospital, 183.
Burrows, H. J. *Book review*, 418.
Osteoporosis (*discussion*), 856.
Perthes' disease (*discussion*), 855.
Burwell, R. G. Cellular immunity and the bone graft, 600.
and Gowland, G. Studies in transplantation of bone.
1—Assessment of antigenicity: serological studies, *814.
2—Changes occurring in lymphoid tissue after homografts and autografts of fresh cancellous bone, *820.
Butler, R. W. Significance of osteochondrosis vertebralis juvenilis in spinal pathology, 202.

C

Caird, D. M. Finger flexor advancement, 188.
Caldwell, R. A., and Collins, D. H. Assessment of vertebral osteoporosis by radiographic and chemical methods post-mortem, *346.
Cameron, D. A. Erosion of epiphysis of rat tibia by capillaries, *590.
Cameron, H. S. Fractures of proximal end of femur in children, 611.
Campbell, C. S. Inserter for small V-type intramedullary nail, *170.
" Lucky breaks," 188.
Campbell Connolly, R. *See* Connolly, R. C.
Capener, N. The accident problem (*symposium*), 182.
Book reviews, 205, 213.
Cubital pain, 398.
Lateral rhachotomy for vertebral biopsy in patients with a suspected tumour, 858.
Surgery of primary osteoarthritis of hip, 856.
Carter, C. O. Genetics in orthopaedics (*editorial*), 217.
Cason, J. S. Burns (*symposium*), 183.
Casuccio, C. Eosinophilic granuloma of bone, 202.
Osteogenesis and delayed consolidation in non-union of long bones, 199.
Cederlöf, S. Follow-up study of osteogenic sarcoma, 186.
Chalmers, J. *Book reviews*, 415, 418.
Cellular immunity and the bone graft (*discussion*), 600.
The halo traction apparatus (*discussion*), 600.
Chapelle, E. H. la. *See* La Chapelle, E. H.
Charnley, J. Arthroplasty of hip by the low-friction technique, 601, 606.
Book review, 414.
Multiple injuries mainly involving lower limbs (*discussion*), 605.
Place of intramedullary nailing in treatment of fractures of femur or tibia, 856.
Surgery of primary osteoarthritis of hip (*discussion*), 856.
and Guindy, A. Delayed operation in open reduction of fractures of long bones, *664.
Charry, R. Resection-angulation of upper end of femur, 202.
Clark, G. L. Congenital dislocation of hip, 189.
Unicameral bone cyst, 188.
Clark, J. M. P. An appraisal of muscle and tendon transfers, 172.
Some aspects of poliomyelitis, 854.
Clark, K. Ganglion of lateral popliteal nerve, *778.
Clarke, H. Osmond-. *See* Osmond-Clarke, H.
Cloke, J. H. *Demonstration*, 615.
Coblentz, J. *See* Forster, E., Mole, L., and Coblentz, J.
Cohen, H., 1st Baron of Birkenhead. Neuropathic joints (*editorial*), 219.
Cohen, S. A. Solitary bone cysts in children, 191.
Collins, D. H. *Book review*, 620.
See also Caldwell, R. A., and Collins, D. H.
Colonna, P. C. Capsular arthroplasty, 194.
Coltart, W. D. Arthroplasty of hip by the low-friction technique (*discussion*), 601.
Book review, 868.
Major injuries: rehabilitation, 182.
Myelography in diagnosis of disc protrusion (*discussion*), 857.
Slipped upper femoral epiphysis (*discussion*), 857.
Commerell, J. J. Treatment of flexor tendon injuries in the hand (*discussion*), 403.
Compere, E. L. Promotion of osteogenesis and prevention of delayed union and pseudarthrosis of long bones, 199.
Connolly, R. C. *Book review*, 866.
Costantini, W. A. Cystic bone lesions, 613.
Cove-Smith, R. The accident problem (*discussion*), 182.
Creer, W. Sayle-. *See* Sayle-Creer, W.
Cregan, J. C. F. Treatment of displaced subcapital fractures of femoral neck in the aged by immediate replacement arthroplasty (*discussion*), 606.
Crock, H. V. Scope and importance of orthopaedic management of haemophiliacs, 172.
Cronquist, S. Myelography in diagnosis of disc protrusion, 857.
Crymble, B. T. Charnley stabilisation of hip, 399.
Cullen, C. H. Paraplegia in extension injuries of spine, 600.

D

Damanski, M. Heterotopic ossification in paraplegia, *286.
Danos, E. Transcervical fractures of femur treated by fixation with Charnley's spring-loaded screw, 399.
David, M. *See* Piollet, J., and David, M.
Davidson, I. W., and Jewett, E. L. Use of vastus intermedius tendon transplant after patellectomy, 612.
Dawson, R. H. Spondylolisthesis (*discussion*), 190.
De Marneffe, R. *See* Marneffe, R. de.
De Wet, I. S. Hazards in orthopaedic surgery, 402.
Define, D. Implantation of fibula in diaphysial bone defects of tibia, 201.
Dega, W. Frequency of congenital hip dysplasia and effectiveness of early treatment, 196.
Derby, A. C. Popliteal cysts, 862.
Devas, M. B. Compression stress fractures in man and the greyhound, *540.
Dommisse, G. F. Functional anatomy of the pelvic joints, 400.
Dooley, B. *See* King, T., and Dooley, B.
Dos Santos, W. R. Intertrochanteric osteotomy and bone graft for non-union of fractures of neck of femur, 202.
Du Toit, G. T. *Discussions*, 401, 403.
Ducharme, F. A. *See* Robichon, J., and Ducharme, F. A.
Duparc, J. *See* Tubiana, R., and Duparc, J.
Duraiswami, P. K. Experimental induction of congenital defects, 203.
Durbin, F. C. Fracture of femoral neck in children (*discussion*), 174.
The halo traction apparatus (*discussion*), 600.
Multiple injuries mainly involving lower limbs (*discussion*), 605.
Durie, E. B., and MacDonald, L. Cryptococcosis of bone, *68.
Dwyer, A. F. *Demonstration*, 615.

* Indicates full article.

Dwyer, F. C. Osteotomy of calcaneum for grossly everted feet, 203.
Treatment of osteochondritic changes complicating treatment of congenital dislocation of hip, 195.

E

Early, P. F. Population studies in Dupuytren's contracture, 604.
Edelstein, J. The Commonwealth Scholarship Act (*discussion*), 173.
Edwards, P. Place of intramedullary nailing in treatment of fractures of femur or tibia, 856.
Eggers, G. W. N. The contact-compression factor in osteogenesis, 199.
Ehrenborg, G. Is Osgood-Schlatter disease caused by fracture? 185.
Elliott, J. K. Cervical spondylosis (*discussion*), 860.
Club foot in the Polynesian, 190.
Ellis, J. *Demonstration*, 615.
Engström, A. Ultrastructure of bone, 185.
Erlacher, P. Congenital dysplasia of hip, 194.
Eskesen, B. *See* Remvig, O., and Eskesen, B.
Esteve, R. Peripheral circulation in poliomyelitis, 203.
Evans, D. Relapsed club foot, *722.
Evans, D. K. Reduction of cervical dislocations, *552.
Evans, E. M. *Book review*, 618.
Evans, E. S. An appraisal of muscle and tendon transfers (*discussion*), 173.
Evans, F. G. Effect of controlled accelerations on stress-strain phenomena in intact vertebral column, 202.
Exner, G. Extensive periosteal change complicating transection paralysis from myelodysplasia, 203.
Secondary operations in treatment of dysplastic hip, 195.
Eyre-Brook, A. L. Multiple injuries mainly involving lower limbs (*discussion*), 605.

F

Fahrni, W. H. Human posture, 610.
Fairbank, T. J. Bone growth following fusion operations (*discussion*), 602.
Book reviews, 211, 868.
Congenital dislocation of hip (*discussion*), 856.
Slipped upper femoral epiphysis (*discussion*), 857.
and Barrett, A. M. Vastus intermedius contracture in early childhood, *326.
Favreau, J. C. Non-tuberculous narrowing of intervertebral disc spaces in children (*discussion*), 610.
Fell, H. B. Action of vitamin A on ground substance of cartilage and bone in culture, 180.
Fergusson, R. S. C. Minor injuries: conditions in Northern Scotland, 183.
Fernandez, A. Lopez-. *See* Lopez-Fernandez, A.
Fitzgerald, H. W. Congenital dysplasia of hip: etiology and treatment, 196.
Orthopaedic and fracture service, Dunedin Hospital and Otago Medical Centre, 200.
Forbes, D. B. Subcortical iliac bone graft in fractures of tibia, *672.
Forster, E., Mole, L., and Coblentz, J. Ligamentous damage in fractures of upper tibia, 192.
Freebody, D. Anterior spinal fusion for lumbar disc lesions (*discussion*), 175.
Freeman, P. A. *See* Stevens, J., *et al.*
Freeman, R. H. The parachute surgical team, 184.
Frew, J. F. M. Familial metaphysial dysplasia, 188.
Fripp, A. T. The problem of the relapsed club foot (*editorial*), 626.
Fry, J. The accident problem: the hospital (*discussion*), 181.

G

Gardemin, H. Early primary chronic osteomyelitis, 201.
Garden, R. S. *Demonstrations*, 607.
Low-angle fixation in fractures of the femoral neck, *647.
Structure and function of proximal end of femur, *576.
Tennis elbow, *100.
Gariépy, R. Surgical treatment of the arthritic knee, 203.
Gayton, W. R. *Demonstrations*, 615
Ghent, W. R. Meralgia paraesthetica, 862.
Ghosh, S. K. Fractured scaphoids, 608.
Gillespie, H. S. Excision of lunate bone in Kienböck's disease, *245.
Gillies, Sir A. Hereditary rickets (*discussion*), 861.
Gillis, L. *Book review*, 414.
Gissane, W. The accident problem: the hospital, 181.
Book review, 207.
Glass, A., and Powell, H. D. W. Traumatic dislocation of hip in children, *29.
Glauber, A. Hyperextension subtrochanteric osteotomy, 202.
Goldhaber, P. Some factors affecting bone resorption in tissue culture, 180.
Golding, F. C. Museum pages:
VI—Spinal cord tumours with gross deformity of spine, *167.
Goodfellow, J. W., and Weaver, J. P. A. Locking of metacarpo-phalangeal joints, *772.
Goodwin, M. A. Primary osteosarcoma of patella, *338.
Gordon, L. H. *See* McPherson, A., Scales, J. T., and Gordon, L. H.
Gowland, G. *See* Burwell, R. G., and Gowland, G.
Grayson, P. C. Metaplasia of synovial membrane, 190.
Osteotomy of calcaneum (*discussion*), 860.
Spontaneous paralysis of posterior interosseous nerve, 860.
Greenfield, B. E. Electromyography of temporomandibular joint, 396.
Griffiths, D. Ll. Myelography in diagnosis of disc protrusion (*discussion*), 857.
Griffiths, H. W. C. Crush injuries of the chest (*editorial*), 623.
Grogono, B. J. S. Bone changes in blood dyscrasia, 612.
Guerra, G. *See* Bonilla, A., and Guerra, G.
Guindy, A. *See* Charnley, J., and Guindy, A.
Guinsburg, R. *See* Petrov, B. A., and Guinsburg, R.
Guntz, E. Storage of bone, 198.
Guthrie, I. An experiment in rehabilitation, 401.

H

Hackenbroch, M. Teaching and training in orthopaedic surgery, 200.
Treatment of paralytic hip, 202.
Haliburton, R. A., Brockenshire, F. A., and Barber, J. R. Avascular necrosis of femoral capital epiphysis after traumatic dislocation of hip in children, *43.
Hall, R. Case of melorheostosis with cutaneous haemangioma and lymphatic vesicles, *335.
Hall, W. H. Control of scoliosis through the growth epiphysis, 858.
Hamilton, A. R. *Demonstrations*, 615.
Lateral epicondylitis of elbow (*discussion*), 614.
Hammer-Jacobsen, E. Inherent risk of ionising radiation, 406.
Hampe, J. F. *See* Kingma, M. J., and Hampe, J. F.

* Indicates full article.

Hancox, N. M., Owen, R., and Singleton, A. Cross-species grafts of deproteinised bone, *152.
Hannay, J. W. Burns (*symposium*), 183.
Hansen, O. M. Influence of exogenic factors on congenital malformations, 406.
Muscle regeneration: incidence of scoliosis and foot deformities after poliomyelitis (*discussion*), 853.
Surgical anatomy and treatment of patients with arthrogryposis, 855.
See also Bro-Rasmussen, F., and Hansen, O. M.
Hargadon, E. J., and Pearson, J. R. Fractures of pelvis involving floor of acetabulum, 605.
Harmon, P. H. Anterior spinal fusion for lumbar disc lesions, 174.
Harmon, P. S. Diagnostic localisation in lumbar disc disease, 400.
Intervertebral disc excision and lumbar spine fusion by a transperitoneal abdominal approach (*discussion*), 401.
Harris, R. I. History of Syme's amputation, 611.
Harrison, M. H. M. Simple prognostic test after fracture of femoral neck, 174.
Harrison, R. M. C. Arthrodesis of joints in growing hand, 863.
Hauberg, G. Congenital dysplasia of hip: formation of acetabular roof, 194.
Hay, B. M. Congenital absence of radius, 189.
Hazlett, J. W. Kissing spines, 863.
Hedley Hall, W. *See* Hall, W. H.
Helfet, A. J. Mid-carpal instability, 403.
Henderson, R. S. Os intermetatarseum, 610.
Hendrix, G. *See* Marneffe, R. de, and Hendrix, G.
Hendry, N. G. C. *See* Mitchell, P. E. G., Hendry, N. G. C., and Billewicz, W. Z.
Hernandez-Ros, A. Use of bone grafts in malposition and non-union of fractures of shafts of long bones, 199.
Hetherington, R. F. *See* Melvin, W. J. S., Hetherington, R. F., and Wollin, D.
Heywood, A. W. B. Recurrent dislocation of patella, *508.
Hicks, J. H. Fractures of forearm treated by rigid fixation, *680.
Hiertonn, T. Reconstructive surgery in peripheral arterial occlusion, 185.
Hněvkovský, O. Progressive fibrosis of vastus intermedius muscle in children, *318.
Hodgkinson, A. *Demonstration*, 615.
Hodgkinson, H. R. T. *Demonstrations*, 615.
Hoets, J. *Demonstration*, 615.
Hogg, T. G. Ulnar neuritis, 614.
Holden, N. T. *See* Nicoll, E. A., and Holden, N. T.
Holdsworth, F. W. *Book review*, 868.
Fractures of spine and their treatment, 401.
Perthes' disease (*discussion*), 855.
Place of intramedullary nailing in treatment of fractures of femur or tibia, 856.
Hornabrook, R. W. *See* Lennon, E. A., Schechter, M. M., and Hornabrook, R. W.
Houlding, R. N., and Matheson, A. T. Intrathecal spinal tumour as a cause of coccydynia, *344.
Houwen, H. van der. Case of neuropathic arthritis caused by indifference to pain, *314.
Howard, R. C. Thoraco-scapular arthrodesis, 175.
Howorth, M. B. Etiology of congenital dysplasia of hip, 197.
Hughes, J. R. The halo traction apparatus (*discussion*), 600.
Hughes, S. Development of jaws and temporomandibular region in tissue culture, 181.
Development of teeth in a chemically defined medium, 181.
Hull, B. L. Scoliosis in binovular twins, *285.
Hunt, J. The accident problem: practice in Russia, 181.
Hutchison, J. Flexor sublimis transfer, 188.
Rupture of calcaneal tendon, 188.

I

Idelberger, K. Leveuf operation in congenital dislocation of hip, 195.
Storage of bone, 198.
Iino, S. Acetabular dysplasia and congenital dislocation of hip, 195.
Isdale, I. *Demonstration*, 861.
The disappearing hip, 860.
Isherwood, I. A radiological approach to the subtalar joint, *566.

J

Jackson, J. P., and Waugh, W. Tibial osteotomy for osteoarthritis of knee, *746.
Jackson Burrows, H. *See* Burrows, H. J.
Jacobsen, E. Hammer-. *See* Hammer-Jacobsen, E.
Jacobsen, L. Irradiation-induced deformities, 407.
James, E. S. Recurrent lumbar disc protrusions, 610.
James, J. I. P. *Book reviews*, 865, 869.
Muscle regeneration: incidence of scoliosis and foot deformities after poliomyelitis (*discussion*), 853.
James, M., and Lassman, L. P. Occult forms of spinal dysraphism, 604.
Jansen, K. Arthrodesis of hip with help of a bone flap, 405.
Pedicle bone graft in hip arthrodesis, 204.
Surgery of primary osteoarthritis of hip (*discussion*), 856.
and Larsen, E. H. J. Congenital dislocation of hip with reference to arthrography, 194.
Jarry, L., and Uhthoff, H. Role of parosteal tissue in post-traumatic osteogenesis of long bones, 612.
Jewett, E. L. *See* Davidson, I. W., and Jewett, E. L.
Johnson, J. T. H. Bone growth following fusion operations, 602.
Jonck, L. M. Mechanical disturbances resulting from lumbar disc space narrowing, *362.
Jones, D. M. Surgical treatment of unstable cervical disc, 858.
Jones, E. A. Familial osteogenic sarcoma, 613.
Incidence of malignant bone tumours in Saskatchewan, 612.
Jones, Sir R. Watson-. *See* Watson-Jones, Sir R.
Judet, J. The study of osteogenesis with its relation to delayed union and pseudarthrosis (*symposium*), 198.
Judet, R. The study of osteogenesis with its relation to delayed union and pseudarthrosis (*symposium*), 197.

K

Kellgren, J. H. Mobility and instability in the cervical spine (*discussion*), 600.
Osteoporosis (*discussion*), 602.
and Lawrence, J. S. Osteoarthritis as seen in random samples of the population, 601.
Kerridge, G. Osteosarcoma, 614.
Kerssemakers, J. G. A. Central dislocation arthroplasty of hip, 192.
Kidd, H. A. Occult forms of spinal dysraphism (*discussion*), 604.
King, A. B. *Demonstrations*, 608.
King, T. Dangers of discography, 615.
and Dooley, B. Late results of McMurray osteotomy, 614.
Kingma, M. J., and Hampe, J. F. Bone heterotransplants, 191.

* Indicates full article.

Kirsten, J. Experimental use of a muscle graft in regeneration of a peripheral nerve, 401.
Kling, K. G. Simple method of bone grafting for non-union and delayed union of fractures, 175.
Knutsson, B. Electromyography as aid to diagnosis of lumbar nerve compression, 857.
Küntscher, G. B. G. Treatment of pseudarthrosis by intramedullary nailing, 198.
Kunkel, M. G. Engelmann's disease, 613.

L

La Chapelle, E. H. Pectoralis major transfer for deltoid paralysis, 192.
Lahz, J. Lateral epicondylitis of elbow, 614.
Laidlaw, A. J. Major injuries: first aid and primary care (*discussion*), 182.
Lange, M. Use of homoplastic and autoplastic bone transplants, 198.
Langenskiöld, A., and Michelsson, J.-E. Experimental progressive scoliosis in rabbit, *116.
Larsen, E. H. J. *See* Jansen, K., and Larsen, E. H. J.
Larsen, J. Schultz-. *See* Schultz-Larsen, J.
Lassen, H. C. A. Life-threatening respiratory paralysis, 853.
Lassman, L. P. *See* James, M., and Lassman, L. P.
Laurin, C. A. " Open sky " bone grafting, 863.
Synovectomy of knee in rheumatoid arthritis, 863.
Lawrence, J. S. *See* Kellgren, J. H., and Lawrence, J. S.
Leak, W. N. The accident problem: the hospital (*discussion*), 181.
Major injuries: first aid and primary care, 182.
Leclerc, J. A. Compression in treatment of non-union of tibial fractures, 863.
Epidural injection of hydrocortisone for proven intervertebral disc protrusions, 863.
Lennon, E. A., Schechter, M. M., and Hornabrook, R. W. Engelmann's disease, *273.
Lepine, E., and Thivierge, A. Primary prosthetic replacement for subcapital fractures of femoral neck, 611.
Liddell, W. A. Cervical spondylosis (*discussion*), 860.
The lumbo-sacral intervertebral foramen, 189.
Lillie, G. J. *Demonstrations*, 398.
Treatment of cerebral palsy, 398.
Lindahl, O. Studies of pain factors, 186.
Lindblom, K. Use of television in hip nailing, 185.
Lindbom, Å. Arteriography and bone tumours, 186.
Linge, B. van. Experimental muscular hypertrophy in the rat, 407.
Lloyd-Roberts, G. C. *See* Spence, A. J., and Lloyd-Roberts, G. C.
Lloyd Williamson, J. C. F. *See* Williamson, J. C. F. Lloyd.
Logan, R. F. L. Major injuries: rehabilitation, 182.
London, P. S. The broken scaphoid bone, *237.
Major injuries: resuscitation and repair of tissues, 182.
Simplicity of approach to treatment of the injured hand, *454.
Lopez-Fernandez, A. Congenital dysplasia of hip (*discussion*), 197.
Louw, J. H. Aorto-iliac occlusive disease, 404.
Lowden, T. G. Minor injuries: anaesthesia and analgesia (*discussion*), 183.
Lowe, H. G. Avascular necrosis after slipping of upper femoral epiphysis, *688.
Lowry, J. Replacement of femoral head with Austin Moore prosthesis, 400.
Results of operation for lumbar disc herniation, 859.
Lutken, P. Development of upper end of femur, 194.
Perthes' disease (*discussion*), 855.

M

McCarroll, H. R. Is congenital dysplasia of hip a forerunner of congenital dislocation? 197.
McDonald, G. A. Fibroma of bone, 863.
Fractures of tibial plateau, 612.
MacDonald, L. *Presidential address, Australian Orthopaedic Association*, 614.
See also Durie, E. B., and MacDonald, L.
McDougall, A. Fracture of neck of femur in childhood, *16.
McFarland, B. L. Congenital absence of radius (*discussion*), 189.
Congenital dislocation of hip, 615.
Congenital dislocation of hip (*discussion*), 603.
Congenital dysplasia of hip (*symposium*), 193, 197.
Grafting bone, 190.
Metaplasia of synovial membrane (*discussion*), 190.
Osteoarthritis as seen in random samples of the population (*discussion*), 601.
Osteoarthritis of hip, 190.
Osteoarthritis of hip (*film*), 614.
Suppurative arthritis of hip in infancy, 191.
Ulnar neuritis (*discussion*), 614.
McGregor, R. M. The accident problem: the general practitioner, 181.
MacIntosh, D. L. Importance of anterior cruciate ligament in relation to tears of medial semilunar cartilage, 612.
McKee, G. K. Arthroplasty of hip by the low-friction technique (*discussion*), 601.
McKenzie, A. B. Congenital absence of radius (*discussion*), 189.
Mackenzie, I. G., and Woods, C. G. Causes of failure after repair of median nerve, *465.
McLean, F. C. The parathyroids and vitamin D in calcium homeostasis, 607.
McMurray, T. B. Recurrent dislocation of shoulder, 402.
McPherson, A., Scales, J. T., and Gordon, L. H. Method of estimating qualitative changes of blood-flow in bone, *791.
and Shaw, N. E. Qualitative method of estimating changes of blood-flow in bone, 601.
Makin, M. Osteogenesis by vesical mucosal transplant, 173.
Marino-Zuco, C. Osteogenesis and pseudarthrosis, 199.
Teaching and training in orthopaedic surgery, 200.
Treatment of congenital dislocation of the hip, 195.
Markowa, J. Acute osteitis of undetermined origin with neuro-infection, *95.
Marneffe, R. de, and Hendrix, G. Congenital talipes equinovarus, 203.
Mårtenson, L. Tomography for ankle fractures, 186.
Matheson, A. T. *See* Houlding, R. N., and Matheson, A. T.
Matheson, J. M. Central sterilisation organisation, 184.
Mau, H. Spontaneous straightening of femoral neck after adduction osteotomy in congenital dysplasia of hip, 194.
Maudsley, R. H., and Arden, G. P. Rheumatoid cysts of calf, *87.
Maxwell, C. M. *Demonstration*, 615.
Mayer, L. Pelvic obliquity in poliomyelitis, 403.
Treatment of flexor tendon injuries in the hand (*discussion*), 403.
Meer, J. van der. Study of the numbers of spastic children needing treatment, 407.
Melvin, W. J. S. Method of hindfoot stabilisation, 610.
Hetherington, R. F., and Wollin, D. Atlanto-axial derangements, 862.

* Indicates full article.

Mercer, Sir W. *Book reviews*, 205, 617, 619, 621, 869.
Edinburgh and early Canadian medicine, 609.
Merle d'Aubigné, R. Organisation of hospital services, 200.
Treatment of non-union in long bones, 198.
Michelsson, J.-E. *See* Langenskiöld, A., and Michelsson, J.-E.
Middleton, R. *Demonstration*, 615.
Miki, I. Radiographic differentiation between acetabular dysplasia and congenital dislocation of hip, 195.
Milgram, J. E. Gradual reduction in congenital dislocation of hip in infancy, 197.
Mills, R. H. Visceral injuries complicating fractures, 399.
Milsom, H. B. C. Club foot in the Polynesian (*discussion*), 190.
Demonstrations, 861.
Review of 118 cases of spondylolisthesis, 190.
Spontaneous paralysis of posterior interosseous nerve (*discussion*), 860.
Mirken, L. Scalenotomy, 402.
Mitchell, G. P. Femoral anteversion (*discussion*), 603.
Mitchell, P. E. G., Hendry, N. G. C., and Billewicz, W. Z. The chemical background of intervertebral disc prolapse, *141.
Moberg, E. Assessment of sensibility in the hand, 186.
Forearm shaft fractures, 186.
Peripheral nerves and hand function (*editorial*), 423.
Moe, J. H. Present concepts in treatment of scoliosis, 611.
Möller, C. Treatment of flexor tendon injuries in the hand (*discussion*), 403.
Mohing, W. Development of hip joint after intertrochanteric osteotomy, 195.
Mole, L. *See* Forster E., Mole, L., and Coblentz, J.
Moller, D. H. *See* Zachariae, L., and Moller, D. H.
Monteiro, J. A. Nova. *See* Nova Monteiro, J. A.
Moreau, J. P. Treatment of fractures of upper end of femur by Pugh nail, 611.
Morris, Sir C. The Commonwealth Scholarship Act, 173.
Mortens, J. Holger Werfel Scheuermann (*obituary*), 394.
Myelography in diagnosis of disc protrusion (*discussion*), 857.
and Sury, B. Chronic respiratory paralysis after poliomyelitis, 854.
Mose, H. *See* Remvig, O., and Mose, H.
Muckart, R. D. De Quervain's disease, 189.
Muller, M. E. Adduction osteotomy for osteoarthritis of hip, 202.
Biomechanical principles in osteosynthesis of pseudarthrosis, 199.
Murdoch, G. Fibrosarcoma of thigh, 187.
Intramedullary nailing and multiple injuries, 188.
Multiple injuries mainly involving lower limbs, 605.
Murley, A. H. G. Surgery of foot in leprosy, 604.
Murr, S. Dislocation of peroneal tendons with marginal fracture of lateral malleolus, *563.

N

Naber, H. G. A. Excision of medial sesamoid of hallux, 191.
Naden, J. R. Arthrodesis of hip (*discussion*), 611.
Nanson, E. M. Vascular complications of gunshot wounds of the extremity, 613.
Napier, J. R. *See* Bowden, R. E. M., and Napier, J. R.
Ness, T. Ulnar nerve compression, 188.
Newman, P. H. Exposure of shoulder joint through axilla and its use in recurrent dislocation (*discussion*), 175.
Nicholson, R. *Demonstration*, 615.
Nicoll, E. A. Myelography in diagnosis of disc protrusion (*discussion*), 857.
Perthes' disease (*discussion*), 855.
Slipped upper femoral epiphysis (*discussion*), 857.
and Holden, N. T. Displacement osteotomy in treatment of osteoarthritis of hip, *50.
Nilsonne, U. Distribution of mineral salts in healing fractures and non-union, 185.
Nisbet, N. W. Biological problems of tissue transplantation, 859.
Nissen, K. I. Morton's metatarsalgia (*discussion*), 604.
Osteoarthritis as seen in random samples of the population (*discussion*), 601.
Surgery of primary osteoarthritis of hip, 856.
Nordin, B. E. C. Osteoporosis (*discussion*), 602.
See also Stevens, J., *et al.*
Nova Monteiro, J. A. New concept on the Monteggia lesion, 201.
Nyholm, K. Steindler's tendon transfer for paralytic elbow, 853.

O

O'Connell, J. E. A. *Book review*, 209.
O'Malley, A. G. Influence of flexor and adductor mechanism in arthritis of hip, 859.
Ortolani, M. Congenital dysplasia of hip: early diagnosis, 194.
Osborne, G. Multiple injuries mainly involving lower limbs (*discussion*), 606.
Osmond-Clarke, H. Accident services, 854.
The Commonwealth Scholarship Act (*discussion*), 173.
Femoral anteversion (*discussion*), 603.
Multiple injuries mainly involving lower limbs (*discussion*), 606.
Myelography in diagnosis of disc protrusion (*discussion*), 857.
Simple method of bone grafting for non-union and delayed union of fractures (*discussion*), 175.
Simple prognostic test after fracture of femoral neck (*discussion*), 174.
Slipped upper femoral epiphysis (*discussion*), 857.
Ottolenghi, C. E. The Chutro-Phemister bone graft, 199.
Outerbridge, R. E. Etiology of chondromalacia patellae, *752.
Owen, R. *See* Hancox, N. M., Owen, R., and Singleton, A.
Oyston, J. K. Madura foot, *259.

P

Paludan, B. Teratomatous effect of vitamin A deficiency in pigs, 406.
Pantazopoulos, T. Extra-articular arthrodesis of subtalar joint, 398.
Parke, W. Club foot in the Polynesian (*discussion*), 190.
Parker, G. Exposure of shoulder joint through axilla and its use in recurrent dislocation, 175.
Parkes, A. Intraneural ganglion of lateral popliteal nerve, *784.
Parry, C. B. Wynn. *See* Wynn Parry, C. B.
Paterson, D. C. Retrosternal dislocation of clavicle, *90.
Patterson, F. P. Results of conservative treatment of fracture of shaft of femur, 611.
Pearson, J. R. *See* Hargadon, E. J., and Pearson, J. R.
Pemberton, P. A. Rotation of acetabular roof in treatment of congenital dislocation of hip, 196.
Petrov, B. A., and Guinsburg, R. Nikolay Nikolaievitch Priorov (*obituary*), 845.

* Indicates full article.

Piggot, J. Traumatic dislocation of hip in childhood, *38.
Pilcher, M. *Book review*, 618.
Piollet, J., and David, M. Ease of reduction of the congenitally dislocated hip in relation to prognosis, 196.
Platt, Sir H. *Book review*, 210.
The Commonwealth Scholarship Act (*discussion*), 173.
Femoral anteversion (*discussion*), 603.
Teaching and training in orthopaedic surgery, 200.
Plewes, L. W. *Book reviews*, 215.
Major injuries: rehabilitation, 182.
Pollen, A. G. Calcareous deposits about metacarpophalangeal joints, *250.
Pollock, G. A. Influence of certain hip muscles upon shape of upper end of femur (*discussion*), 174.
Porter, K. R. D. The accident problem: the administrator, 181.
Powell, H. D. W. Extra centre of ossification for medial malleolus in children, *107.
See also Glass, A., and Powell, H. D. W.
Price, C. H. G. Osteogenic sarcoma: analysis of survival and its relationship to histological grading and structure, *300.
Pridie, K. H., and Alms, M. Medullary nails for tibial fractures, 858.
Proctor, H. Major injuries: first aid and primary care (*discussion*), 182.
Pulvertaft, R. G. Tendon grafts (*editorial*), 421.
Treatment of flexor tendon injuries in the hand, 402.

R

Radulescu, A. Etiology of congenital dislocation of hip, 196.
Ramadier, J. D. Treatment of pseudarthrosis of leg by inter-tibio-fibular grafts, 198.
Rasmussen, F. Bro-. *See* Bro-Rasmussen, F.
Rasmussen, J. Carpal tunnel syndrome, 405.
Rasmussen, K. B. Tendon transfers in forearm and hand for paralytic disorders, 853.
Ratliff, A. H. C. Fracture of femoral neck in children, 174.
Ray, R. D. The study of osteogenesis with its relation to delayed union and pseudarthrosis (*symposium*), 198.
Reilly, M. Approaches to lower limb vascular diseases, 399.
Remvig, O., and Eskesen, B. Some aspects of poliomyelitis, 854.
and Mose, H. Perthes' disease, 855.
Rhydderch, A. I. *Demonstrations*, 615.
Richards, H. Fractures of pelvis involving floor of acetabulum (*discussion*), 605.
Population studies in Dupuytren's contracture (*discussion*), 604.
Rigal, W. M. Tritiated thymidine in studies of chondrogenesis, 180.
Ring, P. A. Influence of nervous system upon growth of bones, *121.
Roaf, R. Bone growth in normal children and its relationship to skeletal maturation (*discussion*), 174.
Giraffe-necked women, *114.
Myelography in diagnosis of disc protrusion (*discussion*), 857.
Paraplegia in extension injuries of spine (*discussion*), 600.
Some aspects of poliomyelitis, 854.
Roberts, G. C. Lloyd-. *See* Lloyd-Roberts, G. C.
Roberts, N. Accident services (*discussion*), 854.
Book review, 409.
Muscle regeneration: incidence of scoliosis and foot deformities after poliomyelitis (*discussion*), 853.
Perthes' disease (*discussion*), 855.
Surgery of primary osteoarthritis of hip (*discussion*), 857.
Roberston, A. R. Internal fixation in forearm fractures, 189.
Robertson, D. E. Aseptic necrosis of distal tibial epiphysis, 613.
Robichon, J., and Ducharme, F. A. Osteotomy of calcaneum in treatment of pes cavus, 610.
Robinson, C. Spinal fusion for tuberculosis, 613.
Rogers, L. Surgical treatment of cervical spondylotic myelopathy, *3.
Ros, A. Hernandez-. *See* Hernandez-Ros, A.
Rose, B. *Demonstrations*, 861.
The disappearing hip (*discussion*), 860.
Rosen, S. von. Congenital dislocation of hip, 855.
Diagnosis and treatment of congenital dislocation of hip in the newborn, 196.
Surgery of primary osteoarthritis of hip (*discussion*), 856.
Rosevelt, P. van. Fractures of proximal end of radius, 407.
Rostrup, O. Teflon as a ligament substitute, 612.
Royle, J. *Demonstration*, 615.

S

Sacks, S. Intervertebral disc excision and lumbar spine fusion by a transperitoneal abdominal approach, 401.
Mid-carpal instability (*discussion*), 403.
St Clair Strange, F. G. *See* Strange, F. G. St C.
Salen, E. Myelography, 185.
Salib, P. Orthopaedic and traumatic skeletal lesions in ancient Egyptians, 203.
Salter, R. B. Innominate osteotomy in treatment of congenital dislocation and subluxation of hip, 196, *518.
Salz, M. *Demonstrations*, 398.
Samuel, E. Radiology of temporo-mandibular joint, 397.
Santos, W. R. dos. *See* Dos Santos, W. R.
Sarkin, T. L. Origin of congenital anomalies and the control of bone form, 401.
Rehabilitative surgery in cerebral palsy, 404.
Saunders, J. Spontaneous paralysis of posterior interosseous nerve (*discussion*), 860.
Savill, D. L. Repair of ruptured calcaneal tendon, 203.
Sayle-Creer, W. Review of Morton's metatarsalgia, 603.
Scaglietti, O. Congenital dysplasia of hip (*symposium*), 193, 197.
Scales, J. T. Arthroplasty of hip by the low-friction technique (*discussion*), 601.
Book review, 416.
See also McPherson, A., Scales, J. T., and Gordon, L. H.
Schechter, M. M. *See* Lennon, E. A., Schechter, M. M., and Hornabrook, R. W.
Schorstein, J. *See* Blockey, N. J., and Schorstein, J.
Schultz-Larsen, J. Influence of genetic factors on congenital deformity, 406.
Schwartz, S. A method of localised fusion of the wrist joint, 404.
Scott, J. C. The accident problem: the hospital (*discussion*), 181.
Femoral anteversion (*discussion*), 603.
Fractures of femoral shaft in adults, 605.
Multiple injuries mainly involving lower limbs (*discussion*), 606.
Slipped upper femoral epiphysis (*discussion*), 857.

* Indicates full article.

Secker Walker, L. *Book review*, 413.
Seddon, H. J. An appraisal of muscle and tendon transfers (*discussion*), 172.
The Commonwealth Scholarship Act (*discussion*), 173.
Femoral anteversion (*discussion*), 603.
The Manchester Ship Canal and the colonial frontier, *425.
Osteoporosis (*discussion*), 602.
The scientific surgeon, *628.
See also Yeoman, P. M., and Seddon, H. J.
Sequers, A. M. *See* Valls, J., Bremner, E., and Sequers, A. M.
Sevitt, S. Major injuries: rehabilitation, 182.
Sharp, I. K. Acetabular dysplasia: the acetabular angle, *268.
Sharp, J. Mobility and instability in the cervical spine (*discussion*), 600.
Shaw, N. E. *See* McPherson, A., and Shaw, N. E.
Shulutka, L. Some problems of bone grafting for loss of bone substance, 204.
Sillar, W. Crushed chest: management of flail anterior segment, *738.
Silva, J. F. Non-union in the shafts of long bones, 198.
Silverstein, E. A. Destructive lesions of spine in Marie-Strümpell arthritis, 863.
Singer, A., and Singer, M. Perthes' disease, 404.
Rotation-varus osteotomy, 404.
Tibialis posterior tendon transfer, 404.
Singer, M. Tibialis posterior transfer in congenital club foot, *717.
See also Singer, A., and Singer M.
Singleton, A. *See* Hancox, N. M., Owen, R., and Singleton, A.
Sissons, H. A. *Book reviews*, 212, 410, 412.
Slee, G. C. European Travelling Scholarship 1960, 848.
Smillie, I. S. The halo traction apparatus (*discussion*), 600.
Upper tibial osteotomy, 187.
Smith, A. DeF. Teaching and training at the New York Orthopaedic Hospital, 201.
Smith, H. G. Rotary mower injuries, 190.
Smith, R. Cove-. *See* Cove-Smith, R.
Smith, W. S. Experimental aspects of so-called congenital dysplasia of hip, 197.
Soeur, R. New technique of surgical immobilisation of bone, 201.
Sofield, H. A. Teaching and training in orthopaedic surgery, 200.
Solomon, L. Bone growth in diaphysial aclasis, *700.
Somerville, E. W. Basic factors in development of congenital dislocation of hip, 195.
Congenital dislocation of hip (*discussion*), 855.
Perthes' disease (*discussion*), 855.
Spence, A. J. European Travelling Scholarship 1959, 176.
and Lloyd-Roberts, G. C. Regional osteoporosis in osteoid osteoma, *501.
Spjut, H. Problems in diagnosis of bone tumours, 186.
Spooner, H. J. *See* Bachynski, B., and Spooner, H. J.
Stanbury, S. W. Bone biopsy in study of metabolic bone disease, 602.
Starr, D. E. Arthrodesis of hip, 611.
Steed, G. R. *Demonstrations*, 398.
Stener, B. Experimental evaluation of hypothesis of ligamento-muscular protective reflexes, 186.
Stening, W. *Demonstration*, 615.
John William Van Rees Hoets (*obituary*), 597.
Stevens, J., *et al.* Incidence of osteoporosis in patients with fracture of neck of femur, 604.
Stewart, I. M. Finger extensor mechanism, 188.
Stirling, R. I. *Book review*, 865.
Stougaard, J. Hereditary factor in osteochondritis dissecans, *256.
Strange, F. G. St C. *Demonstrations*, 184.
Fractures of femoral shaft, 184.
Sury, B. *See* Mortens, J., and Sury, B.
Sutherland, I. D. Funnel chest, 188.

T

Tabatznik, B. *See* Berk, M. E., and Tabatznik, B.
Taine, G. J. Cervical spondylosis, 860.
Taylor, G. W. *Book review*, 866.
Taylor, R. G. Use of bone grafts in malposition and non-union of fractures of shafts of long bones, 199.
Teneff, S. Pathogenesis and treatment of pseudarthrosis of long bones, 200.
Thivierge, A. *See* Lepine, E., and Thivierge, A.
Thompson, H. The halo traction apparatus, 600.
Tidestrom, F. Spine and hip models, 187.
Toit, G. T. du. *See* Du Toit, G. T.
Traub, S. P. The spine in multiple myeloma, 610.
Trevor, D. Femoral anteversion (*discussion*), 603.
Fractures of femoral shaft (*discussion*), 185.
Recurrent dislocation of shoulder (*discussion*), 184.
Trias, A. Effect of persistent pressure on articular cartilage, *376.
See also Trueta, J., and Trias, A.
Trueta, J. *Book reviews*, 211, 212, 416, 618.
Vascular role of reparative osteogenesis, 199.
and Trias, A. Vascular contribution to osteogenesis. IV—Effect of pressure upon epiphysial cartilage of rabbit, *800.
Tubiana, R., and Duparc, J. Prevention of thromboembolic complications in orthopaedic and accident surgery, *7.
Restoration of sensibility in hand by neurovascular skin island transfer, *474.
Tucker, F. R. Non-tuberculous narrowing of intervertebral disc spaces in children, 610.
Tudway, R. C. Radiotherapy for osteogenic sarcoma, *61.
Tupman, G. S. Bone growth in normal children and its relationship to skeletal maturation, 174.
Tyler, R. Hereditary rickets, 861.

U

Uhthoff, H. *See* Jarry, L., and Uhthoff, H.
Urist, M. R. Cellular immunity and the bone graft (*discussion*), 601.
Incidence of osteoporosis in patients with fracture of neck of femur (*discussion*), 605.
Osteoporosis, 602.

V

Valls, J., Bremner, E., and Sequers, A. M. Treatment of chronic osteomyelitis of limbs, 201.
Vandewater, S. L. Fat embolism, 863.
Vertongen, P. Displaced fractures of radial head, 191.
Von Rosen, S. *See* Rosen, S. von.

W

Wade, P. A. The accident surgery of motorways, *634.
Wadsworth, T. G. Traumatic dislocation of hip with fracture of shaft of ipsilateral femur, *47.
Wainwright, D. Septic arthritis of hip in infants, 201.
Slipped upper femoral epiphysis (*discussion*), 857.
Walker, L. Secker. *See* Secker Walker, L.
Walldius, B. Knee arthroplasty with prosthesis, 187.
Ward, T. G. Surgical management of temporomandibular joint derangements, 397.

* Indicates full article.

Wardle, E. N., and Brookes, M. Influence of certain hip muscles upon shape of upper end of femur, 173.
Watson-Jones, Sir R. Bone biopsy in study of metabolic bone disease (*discussion*), 603.
Book review, 869.
Harold Arthur Thomas Fairbank (*obituary*), 595.
Osteoporosis (*discussion*), 602.
Watts, A. *Demonstration*, 615.
Watts, J. C. Missile wounds, 184.
Waugh, W. *See* Jackson, J. P., and Waugh, W.
Weaver, J. P. A. *See* Goodfellow, J. W., and Weaver, J. P. A.
Wells, C. Case of lumbar osteochondritis from the Bronze Age, *575.
Wet, I. S. de. *See* De Wet, I. S.
Wetzenstein, H. Biomechanical studies of the foot. 185.
Wheatley, P. R. Orthopaedics and fracture services in Ghana, 863.
Wheeldon, F. T. Intertrochanteric displacement osteotomy using internal fixation, 399.
Spontaneous fractures of shin in presence of knee deformities, 858.
Wherrett, R. *Demonstration*, 615.
White, A. A. Minor injuries: anaesthesia and analgesia (*discussion*), 183.
Whitlock, R. Facio-maxillary injuries, 400.
Wiberg, G. Electromyography as aid to diagnosis of lumbar nerve compression (*discussion*), 857.
Myelography in diagnosis of disc protrusion (*discussion*), 857.
Slipped upper femoral epiphysis, 857.
Surgery of primary osteoarthritis of hip (*discussion*), 856.
Wiles, P. *Book review*, 209.
Wilkinson, J. A. Femoral anteversion, 603.
Williamson, J. C. F. Lloyd. Introducer for a modified Kessel spline, *170.
Wilson, D. W. Arthrodesis of wrist, 863.
Wilson, E. F. Result after thirty-one years of repair of torn cruciate ligaments, *342.
Wilson, J. N. Fractures of pelvis involving floor of acetabulum (*discussion*), 605.
The halo traction apparatus (*discussion*), 600.
Qualitative method of estimating changes of blood-flow in bone (*discussion*), 601.
Simple method of bone grafting for non-union and delayed union of fractures (*discussion*), 175.
Wilson, J. S. Boyd-. *See* Boyd-Wilson, J. S.
Wilson, R. I. Spinal injuries, 399.
Withers, R. J. W. Samuel Thompson Irwin, Kt. (*obituary*), 844.
Wollin, D. *See* Melvin, W. J. S., Hetherington, R. F., and Wollin, D.
Woods, C. G. Subchondral bone cysts, *758.
See also Mackenzie, I. G., and Woods, C. G.
Woodward, J. Muscle origin release operation for congenital elevation of scapula, 203.
Wright, C. J. E. Long survival in solitary plasmocytoma of bone, *767.
Wright, P. R. *Demonstrations*, 184.
Recurrent dislocation of shoulder, 184.
Wyke, B. D. Neurophysiological aspects of joint function with particular reference to temporomandibular joints, 396.
Wynn Parry, C. B. Electrodiagnosis, *222.

Y

Yeoman, P. M., and Seddon, H. J. Brachial plexus injuries: treatment of the flail arm, *493.

Z

Zachariae, L., and Moller, D. H. Experience with Grice operation for paralytic foot after poliomyelitis, 853.
Zuco, C. Marino-. *See* Marino-Zuco, C.

* Indicates full article.

SUBJECT INDEX

A

Accidents
management of, and general practice (*symposium*), 181.
services, 854.
in Europe, 848.
See also Injuries; Industrial medicine.
Acetabular angle, normal range in adults, *268.
Acetabulum
dysplasia, congenital. *See* Hip dysplasia, congenital.
fractures, 605.
Achilles tendon. *See* Tendo calcaneus.
Aclasis, diaphysial, bone growth in, *700.
Actinomycosis. *See* Madura foot.
Adrenocortical steroids, therapy with
intra-articular, Charcot's arthroplasty complicating (*editorial*), 220.
role in disintegration of rheumatic hip, 860.
See also Hydrocortisone.
Amputations
below-knee, recurrent dislocation of patella after, 509.
Syme's, history of, 611.
upper limb, with arthrodesis of shoulder, for brachial plexus injuries, *493.
Anaesthesia in minor injuries, 183.
Angioma. *See* Haemangioma.
Ankle
arthrodesis, pantalar, one-stage, 610.
fractures, 186.
pain in children associated with extra centre of ossification for medial malleolus, *107.
Ankylosing spondylitis, destructive lesions of spine in, 863.
Aorto-iliac occlusive disease, 404.
Arachnoiditis complicating discography, 615.
Arteries. *See names of arteries. See also* Vascular disorders *etc.*
Arteriography in diagnosis of bone tumours, 186.
Arthritis
chronic
of hip, destruction of femoral head complicating, 860.
treatment, corticosteroids, intra-articular, Charcot's arthropathy complicating (*editorial*), 220.
degenerative. *See* Osteoarthritis.
neuropathic. *See* Charcot's arthropathy.
pyogenic
after steroid injection, 402.
of hip in infancy, 191, 201.
rheumatoid, 861.
knee in
cysts of calf associated with, *87.
flexion deformity, treatment, 203.
treatment, synovectomy, 863.
spine in, cervical, subluxation, 600.
stress fractures of tibia and fibula in, 858.
toes in, claw, treatment, excision of metatarsal heads, 861.
Arthrodesis. *See under names of joints.*
Arthrography. *See under names of joints.*
Arthrogryposis multiplex congenita, 855.
Arthroplasty. *See under names of joints.*
Artificial limb, University of Cape Town design, 403.
Australian Orthopaedic Association
annual meeting, 1960, 613.
joint meeting with New Zealand Orthopaedic Association, 1962, 616.
Automobile injuries, prevention and treatment, *634.
Autoradiography with tritiated thymidine in study of cell division in epiphysial cartilage, 180.

B

Baker's cysts. *See* Synovial cysts.
Belgian Orthopaedic Society. *See* Société Belge d'Orthopédie.
Biceps brachii paralysis, treatment, Steindler's flexorplasty, 853.
Birmingham symposium on accident management and general practice, 1960, 181.
Bladder, urinary, mucous coat of, bone induction by, 173.
Blood
dyscrasias, bone changes in, 612.
flow in bone, qualitative method of estimating changes in, 601, *791.
Blood vessels. *See* Arteries; Haemangioma; Vascular disorders *etc.*; Venous thrombosis.
Bone and bones
blood flow changes in, estimation of, qualitative method, 601, *791.
blood supply adjacent to epiphysial cartilage, effect of pressure on, *800.
carcinoma, fractures in, 862.
changes in blood dyscrasias, 612.
cryptococcosis, *68, *71.
cysts
simple
in children, 191.
pathological fractures near, healing of, 188.
subchondral, *758.
diseases and the general surgeon, 395.
eosinophilic granuloma, 202.
fibroma, desmoplastic, 863.
fibrous dysplasia, polyostotic, hyperparathyroidism in, 613.
grafts and grafting, 190.
for non-union and delayed union of fractures, 204; (*symposium*), 197.
iliac strip grafts, 175, *672.
heterogenous
anorganic, experimental study, *152.
experimental study, 191.
immunity response to, 600.
changes occurring in lymphoid tissue, *820.
serological studies, *814.
" open sky," 863.
preservation, 198
transepiphysial, growth in length of grafts after, 602.
growth
helical growth theory, 401.
influence of nervous system on, *121.
longitudinal
cell division in, *in vitro* study using tritiated thymidine, 180.
in diaphysial aclasis, *700.
of transepiphysial grafts, 602.
pattern in normal children, 174.
See also Epiphysial cartilage; Limbs, lower, length discrepancy.
See also Ossification; Osteogenesis.
infections. *See* Osteitis; Osteomyelitis; *and more specific terms.*
microradiography of fracture healing and non-union, 185.
nerve supply, 133.
Paget's disease, sarcoma in, osteogenic, incidence, geographical, 312.
resorption, some factors affecting, in culture, 180.
structure, molecular, 185.
sarcoma, osteogenic
familial, 613.
follow-up study, 186.

* Indicates full article or appreciable part.

Bone and bones
sarcoma, osteogenic
of patella, primary, *338.
prognosis, 614.
relationship between histological grading and survival rate, *300.
treatment, radiotherapy, *61.
tissue culture of (*symposium*), 180.
tumours
diagnosis, 186.
arteriography, 186.
giant-cell, treatment, resection, 178.
malignant, incidence and survival rate in Saskatchewan, 612.
See also subheadings carcinoma; cysts; eosinophilic granuloma; fibroma; sarcoma.
See also Chordoma; Exostoses; Osteoid osteoma.
See also names of bones.
Bone and Tooth Society, meetings, 180, 396, 607.
Book reviews:
Adams, J. C. Outline of fractures, 3rd edition, 1960, 215.
American Academy of Orthopaedic Surgeons Instructional Course Lectures, Vol. 17, 1960, 867.
Bailey, H. Demonstrations of physical signs in clinical surgery, 13th edition, 1960, 419.
Bauermeister, A. Experimentelle Grundlagen für den Aufbau einer neuen Knochenbank, 1958, 212.
Boos, O. Versorgung von Ohnhändern, 1960, 414.
Burckhart, T. Zur Frage der Unfall- und Berufsbedingten Sehnenscheidentuberkulose, 1959, 418.
Caffey, J. Pediatric x-ray diagnosis, 4th edition, 1961, 869.
Charnley, J. Closed treatment of common fractures, 3rd edition, 1961, 869.
Clinical Orthopaedics, No. 17, 1960, 619.
Colonna, P. C. Principles of orthopaedic surgery, revised edition, 1960, 205.
Cosentino, R. Atlas of anatomy and surgical approaches in orthopaedic surgery, Vol. 2, 1960, 865.
Crisp, E. J. Disc lesions, 1960, 868.
Dayer, L. Le syndrome d'Ellis-van Creveld, 1960, 211.
Ehalt, W. Verletzungen bei Kindern und Jugendlichen, 1961, 409.
Elo, J. O. Effect of subperiosteally implanted autogenous whole-thickness skin graft on growing bone, 1960, 418.
Foote, R. R. Varicose veins, 3rd edition, 1960, 870.
Goff, C. W. Surgical treatment of unequal extremities, 1960, 865.
Grasset, E. J. La coxarthose, 1960, 211.
Guilleminet, M., *ed.* Symposium sur l'os hétéroplastique, 1959, 415.
Hohmann, G., Hackenbroch, M., and Lindemann, K., *eds.* Handbuch der Orthopädie, Bd. 4, Teil 1, 1961, 618.
Holmstrand, K. Biophysical investigations of bone transplants and bone implants, 1957, 412.
International Society of Plastic Surgeons, Transactions of the Second Congress (1959), 1960, 415.
Joseph, J. Man's posture, 1960, 209.
Kaindl, F., *et al.* Lymphangiographie und Lymphadenographie der Extremitäten, 1960, 210.
Key and Conwell's Management of fractures, dislocations and sprains, 7th edition, 1961, 869.
Knese, K.-H. Knochenstruktur als Verbundbau, 1958, 410.
Kulowski, J. Crash injuries, 1960, 207.
Lange, M. Lehrbuch der Orthopädie und Traumatologie, Bd. 1, 1960, 210.
Lièvre, C. A., *ed.* Progressi in ortopedia . . . in onore di Ugo Camera, 1959, 416.
Lindholm, R. Subtalar arthrodesis in poliomyelitis, 1960, 618.
Book reviews (*continued*):
McLarty, M. C. Illustrating medicine and surgery, 1960, 213.
MacQueen, I. A. G. A study of home accidents in Aberdeen, 1960, 418.
Mandarino, M. P. Chemical osteosynthesis in orthopaedic surgery, 1960, 416.
Nachemson, A. Lumbar intradiscal pressure, 1960, 414.
Navés, J., and Rubiés, P. La rodilla, 1959, 212.
Nichols, H. M. Manual of hand injuries, 2nd edition, 1960, 215.
Nilsonne, U. Biophysical investigations of the mineral phase in healing fractures, 1959, 412.
Noordenbos, W. Pain, 1959, 866.
Nuffield Provincial Hospitals Trust. Casualty services and their setting, 1960, 205.
Perkins, G. Orthopaedics, 1961, 617.
Ponlot, R. Le radiocalcium dans l'étude des os, 1959, 413.
Prime, L. M., *ed.* Catalogue of the H. Winnett Orr historical collection, 1960, 621.
Raven, R. W., *ed.* Cancer progress volume 1960, 867.
Rissanen, P. M. Surgical anatomy and pathology of the supraspinous and interspinous ligaments of the lumbar spine, 1960, 413.
Schobinger, R. A. Intra-osseous venography, 1960. 866.
Smillie, I. S. Osteochondritis dissecans, 1960, 409.
Struppler, V., and Witt, N. Verletzungen und Wiederherstellung der oberen Extremitäten, Teil 1, 1961, 866.
Surgery in World War II: Neurosurgery, Vol. 2, 1959, 209.
Valentin, B. Geschichte der Orthopädie, 1961, 868.
Wade, P. A., *ed.* Surgical treatment of trauma, 1960, 618.
Warren, R. Procedures in vascular surgery, 1960, 619.
Watson-Jones, Sir R. Surgery is destined to the practice of medicine, 1961, 868.
Willis, R. A. Pathology of tumours, 3rd edition, 1960, 620.
Zukschwerdt, L., *et al.* Wirbelgelenk und Bandscheibe, 2e Auflage, 1960, 417.
Brachial plexus injuries
complete, irrecoverable, treatment, *493.
shoulder in, abductor paralysis, treatment, transplantation of latissimus dorsi and triceps, 177.
Brachyphalangy with cervical kyphosis and optic atrophy, *77.
Braimbridge, C. V. (*portrait*), 441.
British colonies, medicine and orthopaedics in, *429.
British Commonwealth Scholarship Act, 173.
British Orthopaedic Association
election of Fellows etc., 176, 395, 847.
election of officers, 847.
European Travelling Scholarships, reports of scholars, 176, 848.
meetings, 1960, annual, 172; 1961, spring, 599; 1962, 864.
presidential address, 1961, *628.
Robert Jones Golf Cup, 607.
Robert Jones Medal and Prize, 1961, 396.
Burns, treatment, 183.
Burrows, Harold Jackson, appointment as Chairman of British Editorial Board of the *Journal*, 599.

C

Calcaneal tendon. *See* Tendo calcaneus.
Calcaneum, osteotomy of
for claw foot, 610, 860.
for resistant flat foot, 203.

* Indicates full article or appreciable part.

Calcification, ectopic
about metacarpo-phalangeal joints, *250.
complicating myelodysplasia, 203.
See also Ossification, ectopic.
Calcium homeostasis, parathyroids and vitamin D in, 607.
Calf, rheumatoid cysts of, *87.
Calvé-Legg-Perthes' disease. *See* Hip, osteochondritis.
Canada
medicine in, history of, 609.
Royal College of Physicians and Surgeons, election of Sir Walter Mercer to honorary fellowship, 609.
Canadian Orthopaedic Association, annual meeting, 1960, 610.
Cape Town University artificial limb, 403.
Capener, Norman, election to Council of Royal College of Surgeons of England, 607.
Carcinoma metastatic to bone, fractures in, 862.
Carpal tunnel syndrome, treatment, incision of carpal ligament and reconstruction of tunnel, 405.
Carpus. *See* Wrist.
Cartilage
articular, effect of persistent pressure on, *376.
epiphysial
effect of pressure on, *800.
zone of provisional calcification, erosion by capillaries, *590.
See also Bone growth, longitudinal.
ground substance, action of vitamin A in culture, 180.
semilunar. *See* Menisci.
Cauda equina, ependymoma of, coccydynia due to, *344.
Cerebral palsy
diagnosis, early, 398.
etiology, 407.
foot in, flat, treatment, osteotomy of calcaneum, 203.
reflex patterns in, 861.
treatment, 398.
operative, 179, 203, 404.
Cervical spondylosis, 860.
myelopathy in, treatment, mobilisation of complete spinal cord into an enlarged canal, *3.
treatment, operative, 858.
Cervico-brachial syndrome, treatment, scalenotomy, 402.
Charcot's arthropathy (*editorial*), 219.
due to congenital indifference to pain, *314.
of hip complicating rheumatic disease, 860.
Charnley, John
arthrodesis of hip by central dislocation, results, 399.
arthroplasty of hip by central dislocation, results, 192.
spring-loaded screw for fixation of fractures of femoral neck, results, 399.
Chest
funnel, treatment, operative, results, 188.
injuries, crush (*editorial*), 623.
management of flail anterior segment, *738.
Chondromalacia patellae
etiology, *754.
in recurrent dislocation, 509.
Chordoma
diagnosis, differential, from ependymoma, *167.
of axis vertebra, 615.
Claw foot, treatment, osteotomy of calcaneum, 610, 860.
Claw hand, paralytic, in leprosy, treatment, tendon grafting with radial extensor of wrist as motor, *444.
Claw toes in rheumatoid arthritis, treatment, excision of metatarsal heads, 861.
Club foot
in Polynesians, 190.
recurrent or resistant, treatment (*editorial*), 626.
operative, 179.
osteotomy of cuboid bone, 203.
tendon transplantation, tibialis posterior, *717.
wedge-resection of calcaneo-cuboid joint with soft-tissue release, *722.
Coccydynia due to ependymoma of cauda equina, *344.
Collagen content of intervertebral discs, changes associated with prolapse, *141.
Colonna's capsular arthroplasty of hip for congenital dislocation, 194.
Congenital anomalies
etiology (*symposium*), 405.
helical growth theory, 401.
experimental induction, 203.
of genetic etiology (*editorial*), 217.
Congenital deformities (*demonstration*), 608.
See also under parts of body involved.
Contracture, Dupuytren's, epidemiology, 604.
Corticosteroids. *See* Adrenocortical steroids.
Coxa plana. *See* Hip, osteochondritis.
Coxa valga
in congenital dislocation of hip, treatment, adduction osteotomy, 194, 195.
in pathogenesis of hip dislocation, 173.
Coxa vara
adolescent. *See* Femur, epiphysis, upper, slipping of.
after fracture of femoral neck in children, *21.
Cruciate ligaments
anterior, relation to tears of medial meniscus, 612.
repair of, result after thirty-one years, *342.
Cryptococcosis of bone, *68, *71.
Cuboid bone, osteotomy of, for resistant club foot, 203.
Cysts
epidermoid, intraspinal, in lumbar region of children, *556.
popliteal, 862.
synovial, of calf, in rheumatoid arthritis of knee, *87.
See also Bone cysts; Ganglia.

D

Danish Orthopaedic Association, meetings, 1960, 405.
Deformities
congenital (*demonstration*), 608.
etiology (*symposium*), 405.
helical growth theory, 401.
experimental induction, 203.
of genetic etiology (*editorial*), 217.
fractures in region of—" lucky breaks," 188.
in ancient Egypt, 203.
incidence in Polynesians, 190.
paralytic, etiology, 854.
See also under parts of body involved.
Deltoid muscle paralysis, treatment, transplantation of pectoralis major, 192.
Dermoids. *See* Epidermoids.
Desmoplastic fibroma of bone, 863.
Dewar Orthopaedic Club
foundation, 862.
meeting, 1961, March, 862.
Diaphysial aclasis, bone growth in, *700.
Diaphysial dysplasia, progressive. *See* Engelmann's disease.
Discography, hazards of, 615.
Discs, intervertebral
anatomy and function, *362.
inflammatory disease, non-tuberculous, in children, 610.

* Indicates full article or appreciable part.

Discs, intervertebral
lesions
chemical background of, *141.
diagnosis
electromyography, 857.
myelography, 185, 857.
radiological, 190.
diagnostic localisation in, 400.
etiology
genetic control in mouse, *387.
role of posture in, 610.
mechanical disturbances resulting from lumbar disc space narrowing, *362.
recurrent protrusions, 610.
treatment
arthrodesis, anterior, 174.
by transperitoneal abdominal approach, 401.
hydrocortisone injection, epidural, 863.
operative, 859.
See also Cervical spondylosis; Sciatica.
Dislocations. *See under names of joints.*
Dupuytren's contracture, epidemiology, 604.
Dutch Orthopaedic Association. *See* Nederlandse Orthopaedische Vereniging.
Dysplasia
diaphysial, progressive. *See* Engelmann's disease.
fibrous, polyostotic, hyperthyroidism in, 613.
metaphysial, familial, 188.

E

East Anglian Orthopaedic Club, meeting, 1960, Nov., 608.
Edinburgh and early Canadian medicine, 609.
Education
for less-developed parts of British Commonwealth, 173.
in orthopaedics (*symposium*), 200.
in traumatic surgery, 645.
Egypt, ancient, orthopaedic and traumatic lesions in, 203.
Elbow
pain, 398.
paralysis, flexor, treatment, Steindler's flexorplasty, 853.
tennis, *100.
treatment, 614.
denervation, 615.
ulnar nerve compression at, 614.
treatment, decompression, 188.
Electrodiagnosis, *222.
Electromyography
in diagnosis of lumbar nerve compression, 857.
of temporo-mandibular joint, 396.
Embolism
fat, 863.
pulmonary, complicating orthopaedic and accident surgery, prevention of, *7.
Engelmann's disease, *273.
monostotic, 613.
Eosinophilic granuloma of bone, 202.
Ependymoma
of cauda equina, coccydynia due to, *344.
of spinal cord, *167.
Epicondylitis. *See* Tennis elbow.
Epidermoids, intraspinal, in lumbar region of children, *556.
Epiphysial cartilage
effect of pressure on, *800.
zone of provisional calcification, erosion by capillaries, *590.
See also Bone growth, longitudinal.
Exostoses, osteocartilaginous, multiple hereditary, bone growth in, *700.
Extensor carpi radialis tendon as motor in tendon grafting for claw hand in leprosy, *444.
Extensor carpi radialis brevis tendon lengthening for tennis elbow, *100.
External iliac artery, injury to, during hip surgery, 402.

F

Facio-maxillary injuries, 400.
Fairbank, Sir Harold Arthur Thomas (*obituary*), 595.
Familial metaphysial dysplasia, 188.
Fat embolism, 863.
Femur
deformities of upper end, pathogenesis, role of hip muscles in, 173.
development of upper end, 194.
epiphysis, upper, slipping of (*demonstration*) 608.
diagnosis, new radiographic technique, 187.
necrosis of femoral head after, *688.
treatment, 857.
operative, 861.
wedge osteotomy, 405.
fractures
of head of, associated with dislocation of hip, 179.
of neck of
in children, *16, 174, 611.
in old persons
incidence of, 201.
treatment, arthroplasty, replacement, primary, 606, 611.
necrosis of femoral head after, diagnosis, simple test, 174.
non-union of, treatment, intertrochanteric osteotomy and bone graft, 202.
osteoporosis in patients with, incidence, 604.
treatment
fixation with Charnley's spring-loaded screw, results, 399.
nailing
low-angle nail, 607, *647.
Pugh nail, 611.
use of television in, 185.
of shaft of
knee stiffness after, treatment, quadriceps release, 177.
treatment, 605, 611.
" mobile " traction, 184.
nailing, intramedullary, 856.
delayed, *664.
with dislocation of hip on same side, *47.
head, necrosis
after fractures of femoral neck, 661.
diagnosis, simple test, 174.
in children, *23, 174.
after traumatic dislocation of hip in children, *29, *38, *43.
in slipping of upper epiphysis, 405, *688.
See also Hip, osteochondritis.
neck, anteversion, in congenital dislocation of hip, 603.
osteomyelitis of head of (*demonstration*), 615.
osteotomy
adduction
for coxa valga in congenital dislocation, 194, 195.
for osteoarthritis of hip, 202.
displacement, for osteoarthritis of hip, *50, 856.
internal fixation after, 399
for osteoarthritis of knee, 749.
intertrochanteric, with bone graft, for non-union of fractures of femoral neck, 202.
McMurray's
fixation of, Kessel spline for, modified, with introducer, *170.

* Indicates full article or appreciable part.

Femur
osteotomy
McMurray's, late results, 614.
rotation-varus, 404.
subtrochanteric, hyperextension, for old congenital dislocation of hip, 202.
supracondylar, for recurrent dislocation of patella, results, 512.
wedge, for slipping of upper epiphysis, 405.
resection, proximal, for intractable hip contractures, 863.
resection-angulation of upper end for osteoarthritis of hip, 202.
structure and function of upper end, *576.
Fibroma, desmoplastic, of bone, 863.
Fibrosarcoma of thigh, 187.
Fibrous dysplasia, polyostotic, hyperthyroidism in, 613.
Fibula, fractures
malleolar, marginal, accompanying dislocation of peroneal tendons, *563.
stress, associated with rheumatoid deformity of knee, 858.
Fingers
arthrodesis, in growing hand, 863.
calcareous deposits about metacarpo-phalangeal joints, *250.
congenital abnormalities. *See* Brachyphalangy.
extensor mechanism, 188.
locking of, at metacarpo-phalangeal joint, *772.
tendons, flexor, injuries, treatment, advancement of profundus, 188.
Flat foot
paralytic, in children, treatment, arthrodesis, talo-calcanean, extra-articular, 398.
results, 853.
peroneal spastic, twenty-year follow-up, *734.
resistant, treatment, osteotomy of calcaneum, 203.
Flexor digitorum profundus tendon advancement for injuries of flexor tendons in fingers, 188.
Flexor digitorum sublimis transplantation to thumb, 188.
Fluon. *See* Polytetrafluorethylene.
Foot
arthrodesis. *See* Tarsus, arthrodesis.
bones, accessory, os intermetatarseum, 610.
claw, treatment, osteotomy of calcaneum, 610, 860.
club
in Polynesians, 190.
recurrent or resistant, treatment (*editorial*), 626.
operative, 179.
osteotomy of cuboid bone, 203.
tendon transplantation, tibialis posterior, *717.
wedge-resection of calcaneo-cuboid joint with soft-tissue release, *722.
flat
paralytic, in children, treatment, arthrodesis, talo-calcanean, extra-articular, 398.
results, 853.
peroneal spastic, twenty-year follow-up, *734.
resistant, treatment, osteotomy of calcaneum, 203.
in leprosy, 604.
Madura, *259.
mechanical studies, 185.
Forearm fractures
non-union of, treatment, 186.
of lower end of, in old persons, incidence, 201.
treatment
internal fixation, results, 189.
rigid fixation with lug-plate, *680.
See also under Radius; Ulna.
Fractures
healing of
microradiographic study, 185.
role of parosteal tissue in, 612.
Fractures
in old persons, epidemiology of, 201.
in region of pre-existing deformities—" lucky breaks," 188.
mechanics of, *162.
non-union and delayed union of (*symposium*), 197.
treatment
bone grafting, 204.
iliac strip grafts, 175, *672.
multiple Steinmann pins, 178.
pathological
in metastatic carcinoma, 862.
near simple bone cysts, 188.
stress
compression, in man and the greyhound, *540.
of tibia and fibula associated with rheumatoid deformity of knee, 858.
treatment
nailing, intramedullary
in multiple injuries of lower limb, 188.
inserter for small V-type nail, *170.
Küntscher's, 178.
of femur, 856.
of tibia, 856, 858.
operative, delayed, *664.
wire fixation, new technique, 201.
visceral injuries complicating, 399.
See also under names of bones etc.
Fungus infections. *See* Cryptococcosis; Madura foot.
Funnel chest, treatment, operative, results, 188.

G

Ganglia of lateral popliteal nerve, *778, *784.
Genetics in orthopaedics (*editorial*), 217.
Genu recurvatum after transplantation of tibial tubercle for recurrent dislocation of patella, 513.
Genu valgum in etiology of recurrent dislocation of patella, 509.
Germany, symposium on hand surgery, 1960, 192.
Ghana, orthopaedics in, 863.
Giant-cell tumours of bone, treatment, resection, 178.
Gibson, Alexander, Lecture, University of Manitoba, 1961, 609.
Gigantism, localised, associated with melorheostosis, 335.
Giraffe-necked women, a myth exploded, *114.
Grafting of tissues, biological problems of, 859.
Granuloma, eosinophilic, of bone, 202.
Grice's subtalar arthrodesis for paralytic flat foot, 398.
results, 853.
Growth pattern in normal children, 174.
Gunshot injuries of limbs, vascular complications of, 613.

H

Haemangioma, cutaneous, associated with melorheostosis, *335.
Haemophilia, orthopaedic aspects, 172.
Hallux. *See* Toes, big.
Hallux valgus
associated with os intermetatarseum, 610.
treatment, operative, 178.
" Halo " cervical splint, 600.
Hand
claw, paralytic, in leprosy, treatment, tendon grafting with radial extensor of wrist as motor, *444.
Dupuytren's contracture, epidemiology, 604.
function after nerve injuries, assessment of, *481; (*editorial*), 423.
injuries, treatment, simplicity of approach to, *454.
paralysis, treatment, tendon transplantation, 853.
sensibility
assessment of, 186.

* Indicates full article or appreciable part.

Hand
sensibility, restoration by neurovascular skin island transfer, *474.
surgery, symposium for German-speaking countries, 1960, 192.
tendon transplantation in and about, for paralytic disorders, 853.
tendons
flexor, injuries, treatment, 402.
See also under Tendons.
See also Fingers; Thumb; Wrist.
Hip
arthritis
chronic, disintegration of femoral head complicating, 220, 860.
pyogenic, in infancy, 191, 201.
arthrodesis, 611.
central dislocation operation, results, 399.
pedicle bone graft in, 204, 405.
arthrography in congenital dislocation, 194, 197.
arthroplasty
central dislocation operation, results, 192.
Colonna's capsular, for congenital dislocation, 194.
replacement
by low-friction technique, 601, 606.
primary, for fractures of femoral neck in old persons, 606, 611.
with Austin Moore prosthesis, results, 400.
contracture, intractable, treatment, proximal femoral resection, 863.
dislocation
congenital, 615.
anteversion of femoral neck in, 603.
diagnosis and early treatment, 855.
diagnosis and epidemiology, 603.
old, treatment, osteotomy, subtrochanteric, hyperextension, 202.
treatment
conservative, results, 189.
in Europe, 176.
osteotomy of pelvis, *518.
See also Hip dysplasia, congenital.
paralytic, pathogenesis, coxa valga in, 173.
traumatic
fracture of femoral head associated with, 179.
in children, *29, *38.
necrosis of femoral head after, *43.
with fracture of femoral shaft on same side, *47.
dysplasia, congenital (*symposium*), 193.
diagnosis, measurement of acetabular angle, *268.
in etiology of osteoarthritis, 268.
injuries involving acetabulum, 605.
mechanics, *587.
muscles, influence on shape of upper end of femur, 173.
osteoarthritis, 190.
epidemiology, 601.
etiology, congenital dysplasia, 268.
treatment
arthroplasty, central dislocation operation, results, 192.
muscle release operation, 614, 859.
operative, 856.
osteotomy
adduction, 202.
displacement, *50.
internal fixation after, 399.
McMurray's
fixation of, Kessel spline for, modified, with introducer, *170.
late results, 614.
resection-angulation of upper end of femur, 202.
shelf operation, results, 179.
Hip
osteochondritis, 404, 855, 861.
after congenital dislocation, 193, 194, 195, 197.
treatment, 615.
See also Femur, head, necrosis.
paralysis, treatment, operative, 202.
shelf operation for osteoarthritis, results, 179.
surgery, complications of
injury to external iliac artery, 402.
thrombo-embolic, *8.
Hoets, John William van Rees (*obituary*), 597.
Hospital services
organisation of (*symposium*), 200.
See also Accident services.
Humerus, fractures, epicondylar, 614.
Hydrocortisone injection
epidural, for proven intervertebral disc protrusions, 863.
for tennis elbow, 102.
See also Adrenocortical steroids.
Hyperparathyroidism, 395.
primary (*demonstration*), 608.
See also Renal osteodystrophy.
Hyperthyroidism in polyostotic fibrous dysplasia, 613.
Hypoparathyroidism in infant of mother with hyperparathyroidism, 395.

I

Iliac arteries
external, injury to, during hip surgery, 402.
occlusive disease of, 404.
Ilio-psoas release operation for osteoarthritis of hip, 614, 859.
Industrial medicine, Sir Robert Jones and the Manchester Ship Canal, *425.
Industrial rehabilitation, an experiment in, 401.
Infections
anaerobic, treatment, oxygen under pressure, 407.
prevention of. *See* Sterilisation.
See also more specific terms.
Injuries
due to missiles, 184.
due to rotary mowers, 190.
gunshot, of limbs, vascular complications of, 613.
road, prevention and treatment, *634.
visceral, complicating fractures, 399.
See also Accidents; Burns; Fractures; Industrial medicine; Military surgery; *and under parts of body involved.*
Innominate bone. *See* Pelvis.
International Society of Orthopaedic Surgery and Traumatology, eighth congress, 1960, 193.
Interosseous nerve, posterior, paralysis, spontaneous 860.
Intervertebral discs
anatomy and function, *362.
inflammatory disease, non-tuberculous, in children, 610.
lesions
chemical background of, *141.
diagnosis
electromyography, 857.
myelography, 185, 857.
radiological, 190.
diagnostic localisation in, 400.
etiology
genetic control in mouse, *387.
role of posture in, 610.
mechanical disturbances resulting from lumbar disc space narrowing, *362.
recurrent protrusions, 610.
treatment
arthrodesis, anterior, 174.

* Indicates full article or appreciable part.

Intervertebral discs, lesions,
treatment,
orthrodesis, anterior, by transperitoneal abdominal approach, 401.
hydrocortisone injection, epidural, 863.
operative, 859.
See also Cervical spondylosis; Discography; Sciatica.
Intramedullary nailing
for non-union of fractures, 198.
in multiple injuries of lower limb, 188.
inserter for small V-type nail, *170.
Küntscher's, 178.
of femur, 605, 856.
delayed, *664.
of tibia, 856, 858.
Irish Orthopaedic Club, meetings, 399, 859.
Irwin, Sir Samuel Thompson (*obituary*), 844.
Isotopes, radioactive. *See* Autoradiography.
Italian Orthopaedic Society. *See* Società Italiana di Ortopedia e Traumatologia.

J

Jackson Burrows, Harold. *See* Burrows, Harold Jackson.
Jaws, development in tissue culture, 181.
Joints
cartilage, articular, effect of persistent pressure on, *376.
haemophilic, 172.
laxity of, congenital, in etiology of recurrent dislocation of patella, 509.
neuropathic. *See* Charcot's arthropathy.
neuro-receptor endings in and around, 396.
See also Arthritis; Ligaments; Synovial cysts; Synovial membrane; *and names of joints.*
Jones, Lady, Lecture, Liverpool University, 1961 (*announcement*), 864.
Jones, Sir Reginald Watson-. *See* Watson-Jones, Sir Reginald.
Jones, Sir Robert
and the Manchester Ship Canal, *425.
Golf Cup, 607.
Lecture, Royal College of Surgeons of England, 1960, *425.
Medal and Prize, 1961, 396.
Journal of Bone and Joint Surgery, British Editorial Board, retirement of Sir Walter Mercer as Chairman and appointment of H. Jackson Burrows, 599.

K

Karlen, A. G. (*portrait*), 438.
Kessel spline, modified, with introducer, *170.
Kienböck's disease, treatment, excision of lunate bone, *245.
Knee
arthritis, rheumatoid
cysts of calf in, *87.
flexion deformity in, treatment, 203.
stress fractures of tibia and fibula in, 858.
treatment, synovectomy, 863.
arthroplasty, prosthetic, 187.
cysts of, popliteal, 862.
deformities of epiphysial origin, treatment, osteotomy of tibial table, 187.
flexion, limitation of, in children, due to progressive fibrosis of vastus intermedius, *318, *326.
ligaments
collateral, medial, repair of, 177.
cruciate
anterior, relation to tears of medial meniscus, 612.
repair of, result after thirty-one years, *342.
Knee
ligaments
injuries accompanying fractures of upper end of tibia, 192.
replacement by Teflon, experimental study, 612.
menisci, injuries of medial, relation of anterior cruciate ligament to, 612.
osteoarthritis, treatment
osteotomy of femur, 749.
osteotomy of tibia, *746.
stiffness after fractures of femoral shaft, treatment, quadriceps release, 177.
See also Patella.
Küntscher nailing, 178.
of femur, 605.
delayed, *664.
Kyphosis
adolescent. *See* Scheuermann's disease.
cervical, due to posterior hemivertebrae, *77.

L

Lateral cutaneous nerve compression causing meralgia paraesthetica, 862.
Lateral popliteal nerve, ganglion of, *778, *784.
Leg. *See* Limbs, lower.
Legg-Calvé-Perthes' disease. *See* Hip, osteochondritis.
Leprosy
foot in, 604.
hand in, claw, treatment, tendon grafting with radial extensor of wrist as motor, *444.
Leveuf operation for congenital dislocation of hip, 195.
Ligamento-muscular protective reflexes, hypothesis of, experimental evaluation of, 186.
Ligaments
replacement by Teflon, experimental study, 612.
See also Joints; *and under names of joints.*
Limbs
lower
length discrepancy
after fracture of femoral neck in children, *23.
after nerve and muscle lesions, *121.
associated with melorheostosis, 335.
prosthesis, University of Cape Town design, 403.
upper, paralysis, complete, irrecoverable, due to brachial plexus injuries, treatment, *493.
Little, William John, unveiling of memorial plaque to, 859.
Liverpool University
degree of M.Ch.Orth., examination results, 179.
Lady Jones lecture, 1961 (*announcement*), 864.
McMurray memorial lecture, 395.
" Lucky breaks," 188.
Lumbar nerves, roots of, compression of, diagnosis, electromyography in, 857.
Lumbar puncture, complications, epidermoids, *556.
Lunate bone, osteochondritis, treatment, excision, *245.
Lymphatic vesicles associated with melorheostosis, *335.

M

McMurray Memorial Lecture (Liverpool University), 395.
McMurray's osteotomy
fixation of, Kessel spline for, modified, with introducer, *170.
late results, 614.
Madura foot, *259.
Malleolus, lateral. *See under* Fibula.

* Indicates full article or appreciable part.

Malleolus, medial. *See under* Tibia.
Manchester Ship Canal, Sir Robert Jones and, *425.
Manitoba University, Alexander Gibson lecture, 1961, 609.
Median nerve
and ulnar nerve paralysis in leprosy, treatment, tendon grafting with radial extensor of wrist as motor, *444.
compression in carpal tunnel, treatment, incision of carpal ligament and reconstruction of tunnel, 405.
injuries, repair, causes of failure after, *465.
Medullary nailing. *See* Intramedullary nailing.
Melorheostosis with cutaneous haemangioma and lymphatic vesicles, *335.
Meningioma, misdiagnosed, terminating in spastic quadriplegia and death, 402.
Menisci of knee, injuries of medial, relation of anterior cruciate ligament to, 612.
Meralgia paraesthetica, 862.
Mercer, Sir Walter
election to honorary fellowship of Royal College of Physicians and Surgeons of Canada, 609.
retirement as Chairman of British Editorial Board of the *Journal*, 599.
Metacarpo-phalangeal joints
calcareous deposits about, *250.
locking of , *772.
Metaphysial aclasis, bone growth in, *700.
Metaphysial dysplasia, familial, 188.
Metatarsalgia, Morton's, etiology and pathology, 603.
Microradiography of fracture healing and non-union, 185.
Military surgery, parachute surgical team, 184.
Milwaukee brace, Leeds modification of, 175.
Monteggia fracture-dislocation, prognosis according to type of lesion, 201.
Moore, Austin, hip prosthesis, 400.
Morton's metatarsalgia, etiology and pathology, 603.
Mucopolysaccharides of intervertebral discs, quantitative changes associated with prolapse, *141.
Multiple exostoses, bone growth in, *700.
Multiple myeloma, spine in, 610.
Muscles
grafts of, in nerve repair, 401.
hypertrophy, experimental, 407.
tumours. *See* Rhabdomyosarcoma.
See also Electromyography; Paralysis; *names of muscles; and under names of joints.*
"Museum pages," *167.
Mycetoma pedis. *See* Madura foot.
Mycoses. *See* Cryptococcosis; Madura foot.
Myelodysplasia, periosteal change complicating, extensive, 203.
Myelography, 185.
in diagnosis of disc protrusion, 857.
Myeloma
multiple, spine in, 610.
solitary, long survival in, *767.
Myelopathy. *See under* Spinal cord.
Myositis ossificans. *See* Ossification, ectopic.

N

Nailing, intramedullary
for non-union of fractures, 198.
in multiple injuries of lower limb, 188.
inserter for small V-type nail, *170.
Küntscher's, 178.
of femur, 605, 856.
delayed, *664.
of tibia, 856, 858.
Navicular bone (carpal). *See* Scaphoid bone.
Neck elongation, apparent, in giraffe-necked women, *114.
Nederlandse Orthopaedische Vereniging, meetings, 1959 and 1960, 191.
Neoplasms. *See* Tumours.
Nerves
injuries
hand function after, assessment of, *481; (*editorial*), 423.
repair by muscle graft, experimental, 401.
lateral cutaneous, compression of, causing meralgia paraesthetica, 862.
lateral popliteal, ganglion of, *778, *784.
lesions, electrodiagnosis, *222.
lumbar, roots of, compression of, diagnosis, electromyography, 857.
median
and ulnar, paralysis in leprosy, treatment, tendon grafting with radial extensor of wrist as motor, *444.
compression in carpal tunnel, treatment, incision of carpal ligament and reconstruction of tunnel, 405.
injuries, repair, causes of failure after, *465.
optic, atrophy, congenital, with cervical kyphosis and brachyphalangy, *77.
plantar digital, neuroma of, 603.
posterior interosseous, paralysis, spontaneous, 860.
ulnar, compression at elbow, 614.
treatment, decompression, 188.
Netherlands Orthopaedic Association. *See* Nederlandse Orthopaedische Vereniging.
Neurofibromatosis, relationship to melorheostosis, 337.
New Zealand Orthopaedic Association
annual meetings, 1960, 189; 1961, 859.
joint meeting with Australian Orthopaedic Association, 1962, 616.
Nocardiosis. *See* Madura foot.
Nuffield Orthopaedic Centre, lectures, 1961, Hilary term, 180.

O

Obituaries:
Fairbank, Sir Harold Arthur Thomas, 595.
Hoets, John William van Rees, 597.
Irwin, Sir Samuel Thompson, 844.
Priorov, Nikolay Nikolaievitch, 845.
Scheuermann, Holger Werfel, 394.
Occupational medicine. *See* Industrial medicine *etc.*
Optic nerve atrophy, congenital, with cervical kyphosis and brachyphalangy, *77.
Orthopaedic Travelling Club, first meeting, 1961, 853.
Orthopaedics
education in (*symposium*), 200.
genetics in (*editorial*), 217.
in British colonies, *429.
in Europe, report of British Orthopaedic Association travelling scholar, 176.
in Ghana, 863.
Os calcis. *See* Calcaneum.
Os intermetatarseum, 610.
Os subtibiale, *107.
Osgood-Schlatter's disease, etiology, 185.
Ossification
ectopic
in melorheostosis, 335.
in myelodysplasia, 203.
in paraplegia, clinical study, *286.
See also Calcification, ectopic.
See also Bone growth; Epiphysial cartilage; Osteogenesis.
Osteitis
acute, of undetermined origin, with neuro-infection, *95.
deformans. *See* Paget's disease of bone.
fibrosa cystica. *See* Hyperparathyroidism. *See also* Renal osteodystrophy.

* Indicates full article or appreciable part.

Osteoarthritis
epidemiology, 601.
experimental, effect of persistent pressure on articular cartilage, *376.
of hip, 190.
etiology, congenital dysplasia, 268.
treatment
arthroplasty, central dislocation operation, results, 192.
muscle release operation, 614, 859.
operative, 856.
osteotomy
adduction, 202.
displacement, *50.
internal fixation after, 399.
McMurray's
fixation of, Kessel spline for, modified, with introducer, *170.
late results, 614.
resection-angulation of upper end of femur, 202.
shelf operation, results, 179.
of knee, treatment
osteotomy of femur, 749.
osteotomy of tibia, *746.
of spine, cervical. *See* Cervical spondylosis.
Osteochondritis
dissecans, hereditary factor in *256.
of hip, 404, 855, 861.
after congenital dislocation, 193, 194, 195, 197.
treatment, 615.
See also Femur, head, necrosis.
of lunate bone, treatment, excision, *245.
of spine. *See* Scheuermann's disease.
of tibial epiphysis, lower, 613.
of tibial tubercle, etiology, 185.
Osteochondroma, multiple, bone growth in, *700.
Osteoclastoma, treatment, resection, 178.
Osteogenesis
by vesical mucosal transplant, 173.
in relation to delayed union and pseudarthrosis (*symposium*), 197.
See also Bone growth; Epiphysial cartilage; Ossification.
Osteogenic sarcoma
familial, 613.
follow-up study, 186.
of patella, primary, *338.
prognosis, 614.
relationship between histological grading and survival rate, *300.
treatment, radiotherapy, *61.
Osteoid osteoma, osteoporosis in, regional, *501.
Osteomalacia
after total gastrectomy, 398.
See also Rickets; Renal osteodystrophy.
Osteomyelitis
chronic
primary, 201.
treatment, 201.
of femoral head (*demonstration*), 615.
of spine. *See* Intervertebral discs, inflammatory disease.
See also more specific terms.
Osteoporosis, 602, 856.
accompanying fractures of femoral neck, incidence 604.
after nerve and muscle lesions, *121.
etiology, 395.
in etiology of fractures in old persons, 201.
measurement of, 604.
regional, in osteoid osteoma, *501.
spinal, assessment by radiographic and chemical methods post-mortem, *346.
Osteotomy. *See under names of bones.*
Oxford University, Nuffield Orthopaedic Centre, lectures, 1961, Hilary term, 180.

P

Paget's disease of bone, sarcoma in, osteogenic, incidence, geographical, 312.
Pain
factors, experimental study, 186.
indifference to, congenital (*editorial*), 220.
Charcot's arthropathy due to, *314.
Parachute surgical team, 184.
Paralysis
deformities following, etiology, 854.
due to lower motor neuron lesions, bone growth disturbances in, *121.
electrodiagnosis, *222.
treatment, tendon transplantation, 172.
See also Cerebral palsy; Poliomyelitis; *and under parts of body involved.*
Paraplegia
after poliomyelitis, rehabilitation, 854.
due to myelodysplasia, extensive periosteal change complicating, 203.
ectopic ossification in, clinical study, *286.
in hyperextension injuries of spine, mechanism of, 600.
Parathyroid glands
in calcium homeostasis, 607.
See also Hyperparathyroidism.
Parkinson, James, unveiling of memorial plaque to, 859.
Patella
chondromalacia of, etiology, *754.
dislocation, recurrent, pathology and treatment, *508.
elevation of, in children, due to progressive fibrosis of vastus intermedius, *318.
sarcoma, osteogenic, primary, *338.
Patellectomy, repair of resulting gap by transplantation of vastus intermedius tendon, 612.
Pectoralis major transplantation for paralysis of deltoid, 192.
Pectus excavatum, treatment, operative, results, 188.
Pelvis
fractures, involving acetabulum, 605.
joints of, functional anatomy, 400.
obliquity of, in poliomyelitis, 403.
osteotomy of, for congenital dislocation and subluxation of hip, *518.
Peripheral nerves. *See* Nerves.
Peroneal spastic flat foot, twenty-year follow-up, *734.
Peronei tendons, dislocation, with marginal fracture of lateral malleolus, *563.
Perry-Nickel "halo" splint, 600.
Perthes' disease. *See* Hip, osteochondritis.
Pes cavus. *See* Claw foot.
Pes planus. *See* Flat foot.
Pes valgus. *See* Flat foot.
Phemister bone graft, 199.
Plantar digital neuroma, 603.
Plasmacytoma. *See* Myeloma.
Plastics. *See* Polytetrafluorethylene.
Platt, Sir Harry, presentation of own portrait to, by British Orthopaedic Association, 176.
Poliomyelitis
foot in, flat, treatment, arthrodesis, talocalcaneum, extra-articular, results, 853.
leg length discrepancy in, *121.
muscle recovery after, 853.
paraplegia in, rehabilitation, 854.
pelvic obliquity in, 403.
respiratory paralysis in, 853, 854, 857.
scoliosis after, incidence, 853.

* Indicates full article or appreciable part.

Poliomyelitis
shoulder in, deltoid paralysis, treatment, transplantation of pectoralis major, 192.
vascular disorders in, 203.
Polysaccharides. *See* Mucopolysaccharides.
Polytetrafluorethylene
as ligament substitute, experimental study, 612.
in hip arthroplasty, 601, 606.
Popliteal cysts, 862.
Posterior interosseous nerve, paralysis, spontaneous, 860.
Posture, role in etiology of degenerative changes in spine, 610.
Priorov, Nikolay Nikolaievitch (*obituary*), 845.
Progressive diaphysial dysplasia. *See* Engelmann's disease.
Prostheses, lower limb, University of Cape Town design, 403.
Psoas major. *See* Ilio-psoas.
Pubic symphysis, physiology and pathology, 400.
Pugh nail for fractures of femoral neck, 611.
Pulmonary embolism complicating orthopaedic and accident surgery, prevention of, *7.

Q

Quadriceps femoris
release operation for knee stiffness after fractures of femoral shaft, 177.
See also Vastus intermedius.

R

Radiation injury in etiology of congenital deformities, 406, 407.
Radioactive isotopes. *See* Autoradiography.
Radius
absence of, congenital, 189.
fractures
of upper end of, 407.
displaced, 191.
See also under Forearm.
von Recklinghausen's disease of bone. *See* Hyperparathyroidism. *See also* Renal osteodystrophy.
von Recklinghausen's disease of nerve. *See* Neurofibromatosis.
Rehabilitation, industrial, an experiment in, 401.
Renal failure, acute, complicating crush injuries of chest, 623.
Renal osteodystrophy, glomerular, 602.
Research, surgical, *628.
Rhabdomyosarcoma, 398.
Rheumatoid arthritis, 861.
knee in
cysts of calf associated with, *87.
flexion deformity, treatment, 203.
treatment, synovectomy, 863.
spine in, cervical, subluxation, 600.
stress fractures of tibia and fibula in, 858.
toes in, claw, treatment, excision of metatarsal heads, 861.
Rickets
hereditary, 861.
See also Osteomalacia; Renal osteodystrophy.
Road injuries, prevention and treatment, *634.
Robertson, E. L. R. (*portrait*), 439.
Royal College of Physicians and Surgeons of Canada, election of Sir Walter Mercer to honorary fellowship, 609.
Royal College of Surgeons of England
election of Norman Capener to Council, 607.
Robert Jones lecture, 1960, *425.
Watson-Jones lecture, 1960, *634.

S

Sacro-iliac joint, disorders in and around, 400.
Sarcoma
osteogenic
familial, 613.
follow-up study, 186.
of patella, primary, *338.
prognosis, 614.
relationship between histological grading and survival rate, *300.
treatment, radiotherapy, *61.
See also Fibrosarcoma; Rhabdomyosarcoma; *and under names of tissues.*
Scalenus anterior syndrome, treatment, scalenotomy, 402.
Scaphoid bone (carpal), fractures, 608.
case against pessimism, *237.
non-union of, treatment, operative, 179.
Scapula, high, congenital, treatment, muscle origin release, 203.
Scheuermann, Holger Werfel (*obituary*), 394.
Scheuermann's disease
lumbar, case from Bronze Age, *575.
pathology, 202.
Sciatica
due to encroachment on lumbo-sacral intervertebral foramen, 189.
See also Intervertebral discs, lesions.
Scientific research in surgery, *628.
Scoliosis
congenital, 202.
experimental, in rabbit, *116.
idiopathic, in binovular twins, *285.
paralytic, after poliomyelitis, incidence, 853.
treatment, 611.
arthrodesis of spine with iliac grafts first implanted subcutaneously in thigh, 204.
Milwaukee brace, Leeds modification of, 175.
stapling, results, 858.
Scottish Orthopaedic Club, annual meeting, 1960, 187.
Semilunar cartilages. *See* Menisci.
Sesamoid bones of big toe, excision of medial, results, 191.
Shoulder
approach to, axillary, 175.
arthrodesis
thoraco-scapular, 175.
with amputation through arm, for brachial plexus injuries, *493.
dislocation, recurrent, 184, 402.
treatment, 177.
paralysis
abductor, in brachial plexus injuries, treatment, transplantation of latissimus dorsi and triceps, 177.
deltoid, treatment, transplantation of pectoralis major, 192.
See also Scapula.
Skeleton. *See* Bone and bones.
Skin grafting, tubed pedicle based on inferior epigastric artery, 179.
Società Italiana di Ortopedia e Traumatologia, annual congress, 1960, 193.
Société Belge d'Orthopédie, meeting, 1960, with Nederlandse Orthopaedische Vereniging, 191.
Société Internationale de Chirurgie Orthopédique et de Traumatologie, eighth congress, 1960, 193.
South African Orthopaedic Association, annual congress, 1960, 400.
South-East Metropolitan Regional Orthopaedic Club, meetings, 1960, 184.
South-West Orthopaedic Club, meetings, 1960, 185, 397; 1961, 858.
Spina bifida occulta, 604.

* Indicates full article or appreciable part.

Spinal cord
congenital abnormalities. *See* Myelodysplasia.
involvement in cervical spondylosis, treatment, mobilisation of complete cord into an enlarged canal, *3.
tumours with gross deformity of spine, *167.
See also Cauda equina; Paraplegia.
Spine
arthrodesis
anterior
by transperitoneal abdominal approach, 401.
for tuberculosis, 613.
lumbar, 174.
in children, growth in length of grafts after, 602.
with full-thickness graft from iliac crest, 202.
with iliac grafts first implanted subcutaneously in thigh, 204.
biopsy, lateral rhachotomy for, 858.
cervical
atlanto-axial derangements, 862.
congenital kyphosis due to posterior hemivertebrae, *77.
dislocations, reduction of, *552.
elongation, apparent, in giraffe-necked women, *114.
splint, " halo," 600.
subluxation, in rheumatoid arthritis, 600.
chordoma of axis vertebra, 615.
congenital anomalies due to abnormal development of vertebral body, *83.
fractures, 401.
in multiple myeloma, 610.
injuries, 399.
hyperextension, with paraplegia, necropsy findings, 600.
" kissing," 863.
osteochondritis. *See* Scheuermann's disease.
osteoporosis, assessment by radiographic and chemical methods post-mortem, *346.
stresses and strains in, due to high acceleration, 202.
tuberculosis, treatment, arthrodesis, anterior, 613.
tumours, epidermoid, in lumbar region of children, *556.
See also Intervertebral discs; Kyphosis; Scoliosis.
Spondylarthritis, 610.
Spondylitis, ankylosing, destructive lesions of spine in, 863.
Spondylolisthesis, 190, 861.
treatment, arthrodesis, anterior, 174.
Spondylosis, cervical, 860.
myelopathy in, treatment, mobilisation of complete spinal cord into an enlarged canal, *3.
treatment, operative, 858.
Staphylococcal infections. *See* Arthritis, pyogenic; Osteomyelitis.
Steindler's flexorplasty of elbow, 853.
Sterilisation service, central, at Cambridge Military Hospital, Aldershot, 184.
Sterno-clavicular joint, dislocation, posterior, *90.
Steroids, adrenocortical, therapy with
intra-articular, Charcot's arthropathy complicating (*editorial*), 220.
role in disintegration of rheumatic hip, 860.
See also Hydrocortisone.
" Stiff man " syndrome, 398.
Subtalar joint. *See* Talo-calcanean joint.
Surgery, research in, *628.
Syme's amputation, history of, 611.
Symphysis pubis, physiology and pathology, 400.
Synovectomy. *See under names of joints.*
Synovial cysts of calf in rheumatoid arthritis of knee, *87.
Synovial membrane, metaplasia of, 190.

T

Talipes. *See* Club foot.
Talo-calcanean joint
arthrodesis, extra-articular, for paralytic flat foot, 398.
results, 853.
radiological approach to, *566.
Talus accessorius, *107.
Tarsus
arthrodesis
pantalar, one-stage, 610.
without excision of joint spaces, 610.
synostosis, incidence in peroneal spastic flat foot, *734.
See also names of tarsal bones and joints.
Teeth, development in tissue culture, 181.
Teflon. *See* Polytetrafluorethylene.
Television in hip nailing, 185.
Temporo-mandibular joint (*symposium*), 396.
Tendo calcaneus, rupture, 188, 203.
Tendons
grafting (*editorial*), 421.
new technique of anastomosis, *444.
transplantation, 172, 854.
See also names of tendons, and under parts of body.
Tennis elbow, *100.
treatment, 614.
denervation, 615.
Thoracic outlet syndrome, treatment, scalenotomy, 402.
Thoraco-scapular arthrodesis, 175.
Thorax. *See* Chest.
Thrombo-embolism complicating orthopaedic and accident surgery, prevention of, *7.
Thumb paralysis, treatment, transplantation of flexor sublimis, 188.
Thymidine, tritiated, in *in vitro* study of cell division in epiphysial cartilage, 180.
Thyroid gland. *See* Hyperthyroidism.
Tibia
defects of shaft of, treatment, implantation of fibula, 201.
epiphysis, lower
extra centre of ossification for medial malleolus, *107.
osteochondritis, 613.
fractures
mechanics of, *162.
non-union and delayed union of, treatment
bone grafting
inter-tibio-fibular, 198.
subcortical iliac, *672.
compression in, 863.
fibular by-pass operation, 404.
of plateau of, 612.
of upper end of, with ligamentous damage, 192.
stress, associated with rheumatoid deformity of knee, 858.
treatment
in Padua, 176.
nailing, intramedullary, 856, 858.
osteotomy
for osteoarthritis of knee, *746.
of upper end of, for deformities of knee, 187.
torsion of, in etiology of recurrent dislocation of patella, 509.
tubercle of, osteochondritis, etiology, 185.
Tibialis posterior transplantation, 404.
for club foot, *717.
Tissue transplantation, biological problems of, 859.
Toes
big
sesamoid bones of, excision of medial, results, 191.
See also Hallux valgus.
claw, in rheumatoid arthritis, treatment, excision of metatarsal heads, 861.

* Indicates full article or appreciable part.

Torulosis. *See* Cryptococcosis.
Training. *See* Education.
Transplantation of tissues, biological problems of, 859.
Trauma. *See* Injuries.
Tritiated thymidine in *in vitro* study of cell division in epiphysial cartilage, 180.
Tropical medicine in British colonies, *429.
Tuberculosis of spine, treatment, arthrodesis, anterior, 613.
Tumours
giant-cell, of bone, treatment, resection, 178.
See also more specific terms, and under names of tissues and parts of body.
See also Cysts.

U

Ulna, fractures
Monteggia, prognosis according to type of lesion, 201.
See also under Forearm.
Ulnar nerve
and median nerve paralysis in leprosy, treatment, tendon grafting with radial extensor of wrist as motor, *444.
compression at elbow, 614.
treatment, decompression, 188.

V

Vascular disorders
in poliomyelitis, 203.
occlusive
aorto-iliac, 404.
treatment, reconstructive surgery, 185.
Vascular injuries complicating gunshot wounds of limbs, 613.
Vastus intermedius
fibrosis, progressive, in children, *318, *326.
tendon transplantation to fill gap after patellectomy, 612.
Venous thrombosis and embolism complicating orthopaedic and accident surgery, prevention of, *7.
Vitamin A
action on cartilage ground substance in culture, 180.
deficiency in etiology of congenital deformities, experimental study, 406.
Vitamin D in calcium homeostasis, 607.
Von Recklinghausen's disease of bone. *See* Hyperparathyroidism. *See also* Renal osteodystrophy.
Von Recklinghausen's disease of nerve. *See* Neurofibromatosis.

W

Watson-Jones, Sir Reginald, Lecture, Royal College of Surgeons of England, 1960, *634.
Wilkinson, J. A., awarded Robert Jones Medal and Prize, 396.
Wrist
arthrodesis, 863.
localised, 404.
instability, intercarpal, 403.
median nerve compression at. *See* Carpal tunnel syndrome.
nerve injuries at, median, repair, causes of failure after, *465.
See also names of carpal bones.

Y

Yugoslav Association of Orthopaedic and Traumatological Surgeons, foundation of, 864.

* Indicates full article or appreciable part.

CONTRIBUTORS—NOVEMBER 1961

Professor ROLAND BARNES, F.R.C.S., Orthopaedic Department, Western Infirmary, Glasgow, W.1, Scotland.

GEORGE BONNEY, Esq., M.S., F.R.C.S., 83 Harley Street, London, W.1, England.

J. G. BONNIN, Esq., F.R.C.S., 129 Harley Street, London, W.1, England.

G. T. F. BRADDOCK, Esq., F.R.C.S., King Edward Memorial Hospital, Ealing, London, W.13, England.

R. GEOFFREY BURWELL, Esq., M.D., F.R.C.S., Department of Anatomy, The School of Medicine, Leeds, 2, England.

JOHN CHARNLEY, Esq., F.R.C.S., Centre for Hip Surgery, Wrightington Hospital, nr. Wigan, Lancashire, England.

KENNETH CLARK, Esq., F.R.C.S.Ed., 27 Boon Avenue, Penkhull, Stoke-on-Trent, Staffordshire, England.

W. D. COLTART, Esq., F.R.C.S., 58 Harley House, Regent's Park, London, N.W.1, England.

R. CAMPBELL CONNOLLY, Esq., F.R.C.S., Royal National Orthopaedic Hospital, 234 Great Portland Street, London, W.1, England.

DILLWYN EVANS, Esq., F.R.C.S.Ed., 25 Cathedral Road, Cardiff, Wales.

T. J. FAIRBANK, Esq., F.R.C.S., 10 Cranmer Road, Cambridge, England.

Dr DAVID B. FORBES, M.D., 6575 Victoria Avenue, Apartment 18, Montreal, Canada.

A. T. FRIPP, Esq., F.R.C.S., 25 Upper Wimpole Street, London, W.1, England.

R. S. GARDEN, Esq., M.Ch.Orth., F.R.C.S.Ed., 32 Ribblesdale Place, Preston, England.

J. W. GOODFELLOW, Esq., F.R.C.S., Radcliffe Infirmary, Oxford, England.

Dr LARRY GORDON, M.D., United States Naval Hospital, Memphis, Tennessee, United States of America.

G. GOWLAND, Esq., B.Sc., Ph.D., Department of Zoology, University College, Gower Street, London, W.C.1, England.

Dr H. W. C. GRIFFITHS, D.A., F.F.A.R.C.S., Royal Infirmary, Edinburgh, Scotland.

ADLY GUINDY, Esq., M.S., 18 Koubba Street, Heliopolis, Cairo, Egypt.

J. H. HICKS, Esq., M.Ch.Orth., F.R.C.S., Birmingham Accident Hospital, Birmingham, 15, England.

J. P. JACKSON, Esq., F.R.C.S., Birchholt, Mansfield Road, Linby, Nottinghamshire, England.

Professor J. I. P. JAMES, M.S., F.R.C.S., Department of Orthopaedic Surgery, Royal Infirmary, Edinburgh, Scotland.

H. G. LOWE, Esq., F.R.C.S., Staffordshire General Infirmary, Stafford, England.

Dr ANGUS MCPHERSON, M.B., Ch.B., M.R.C.P., Institute of Orthopaedics, Royal National Orthopaedic Hospital, Brockley Hill, Stanmore, Middlesex, England.

Sir WALTER MERCER, F.R.C.S.Ed., Bidston, 7 Easter Belmont Road, Edinburgh, 12, Scotland.

Dr R. E. OUTERBRIDGE, M.D., F.R.C.S.(C.), F.A.C.S., 713 Columbia Street, New Westminster, British Columbia, Canada.

ATHOL PARKES, Esq., F.R.C.S.Ed., 72 Lauderdale Gardens, Glasgow, W.2, Scotland.

Dr JOHN T. SCALES, M.R.C.S., L.R.C.P., Institute of Orthopaedics, Royal National Orthopaedic Hospital, Brockley Hill, Stanmore, Middlesex, England.

H. J. SEDDON, Esq., *C.M.G.*, D.M., F.R.C.S., Institute of Orthopaedics, Royal National Orthopaedic Hospital, 234 Great Portland Street, London, W.1, England.

W. SILLAR, Esq., *M.C.*, F.R.F.P.S.G., F.R.C.S.Ed., Accident and Orthopaedic Service, Southern General Hospital, Glasgow, Scotland.

MARTIN SINGER, Esq., F.R.C.S., 909 Norwich House, Heerengracht, Cape Town, South Africa.

L. SOLOMON, Esq., F.R.C.S., Royal National Orthopaedic Hospital, 234 Great Portland Street, London, W.1, England.

R. I. STIRLING, Esq., F.R.C.S.E., 19 Cluny Drive, Edinburgh, 10, Scotland.

Professor G. W. TAYLOR, M.S., F.R.C.S., Surgical Professorial Unit, St Bartholomew's Hospital, London, E.C.1, England.

Dr ANTONI TRIAS, M.D., Department of Orthopaedic Surgery, University of Illinois, 840 South Wood Street, Chicago, Illinois, United States of America.

Professor J. TRUETA, M.D., F.R.C.S., Nuffield Orthopaedic Centre, Oxford, England.

Professor PRESTON A. WADE, A.B., M.D., F.A.C.S., 898 Madison Avenue, New York City, United States of America.

Sir REGINALD WATSON-JONES, M.Ch.Orth., F.R.C.S., 82 Portland Place, London, W.1, England.

W. WAUGH, Esq., M.Chir., F.R.C.S., Hill House, Lambley Lane, Lowdham, Nottinghamshire, England.

J. P. A. WEAVER, Esq., F.R.C.S.Ed., Royal Infirmary, Newcastle upon Tyne, England.

Dr C. G. WOODS, M.B., Ch.B., Department of Pathology, The School of Medicine, Leeds, 2, England.

Dr C. J. E. WRIGHT, M.D., Department of Pathology, The School of Medicine, Leeds, 2, England.